INTERNAL MEDICINE

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*WITH 109 ILLUSTRATIONS IN THE TEXT AND
7 COLORED PLATES*

To

HENRY FAIRFIELD OSBORN, LLD., D.Sc.

PROFESSOR OF ZOOLOGY IN COLUMBIA
UNIVERSITY, AND FORMERLY PROFESSOR OF
COMPARATIVE ANATOMY IN PRINCETON
UNIVERSITY, THIS BOOK IS DEDICATED IN
GRATEFUL RECOGNITION OF THE FACT THAT
FROM HIM THE WRITER FIRST LEARNED
THE JOY OF INDEPENDENT STUDY AND
INVESTIGATION

PREFACE

DOUBTLESS there has rarely been a teacher of medicine who has been satisfied with the books which his students have had to use. The large treatises are too long, too rich in information for those entering upon the study of medicine, while many of the shorter works are mere catalogues of facts. It has seemed to the writer, after many years' teaching, that there was a distinct need both among students and practitioners of a work which should give in compact form the more important facts of the subjects included in the domain of internal medicine. Just as the modern builder rears his frame-work of steel and upon this suspends the walls, the floors and the ornamentation of his building, so the student must outline his subject in his mind, and later add the details. The beauty of the building will depend upon the finish, its strength must lie in the frame-work. The purpose of the present work is to supply the frame-work of internal medicine.

The title has been chosen in recognition of the need of a term which shall denote those subjects left of the older '' practice of medicine '' when all the specialties have been subtracted from it, those subjects which are, after all, the fundamentals of our study.

The arrangement of the subjects has been dictated by the wish to put the simpler first, and to present something of a logical order of study. The placing of some subjects, such as that of small-pox, among diseases due to animal parasites, is questionable, but in such instances it has seemed well to follow the best leading we have at present, although we may be uncertain of its correctness. The final classification of many of the familiar diseases must long remain doubtful. Syphilis has for years been regarded as surely a bacterial disease and classified accordingly. The recent studies apparently prove the specific organism to belong to the lower orders of the animal kingdom. At any time further investigation may restore the disease to its older position. In like manner the specific agents of scarlet fever, measles, and other exanthemata are assumed to be bacteria, and these diseases are placed accordingly. The eager investigations now going on in so many laboratories may any day give one or more of these diseases a new position. We must act upon our present knowledge, with full consciousness that much of it may tomorrow require revision.

The lack of illustration in the current works on our subject has been very striking in this day when photography is so readily available and is so great an aid in the presentation of any subject. It has been a source of pleasure that the publishers have wished to make full use of this aid

and have so heartily assisted the author in his efforts to this end. To the many friends who have loaned their treasures to him, the author renews his thanks. The effort has been made to render due recognition for such assistance.

Especial thanks are due to my friends, Dr. Walter B. James for aid in writing the chapter on Cardiac Arrhythmia, and Dr. Lewis A. Conner for much valuable aid in the preparation of the text.

<div align="right">THE AUTHOR.</div>

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INTERNAL MEDICINE

I.

DISEASES OF THE RESPIRATORY TRACT

DISEASES OF THE NOSE

ACUTE RHINITIS
(Acute Coryza. Acute Nasal Catarrh)

Definition.—An acute inflammation of the nasal mucous membrane.

Etiology.—The affection is common at all ages, especially during the winter and spring months. Exposure to cold and wet is the most frequent antecedent. The various bacteria to be found in the nose are believed to have a part in the causation. The affection is frequently transmitted from one member of a household to the others and may become epidemic. An acute rhinitis belongs to the invasion of measles.

Morbid Anatomy.—The nasal mucous membrane is congested and swollen, sometimes to such a degree as to block the nares; it is dry at the beginning, later covered by a free secretion of mucus or muco-pus.

Symptoms.—These are general and local. (1) General: There is usually more or less malaise, muscular soreness or pains in various parts of the body, with or without fever. The temperature may rise to 100° or 102°, and the pulse be quickened correspondingly. (2) Local: Sneezing may mark the onset. The nose becomes dry and uncomfortable, and in the course of a few hours the nares are obstructed by the swelling and a free mucous secretion is established. This secretion often irritates the nares and upper lip, causing an eczema. Usually there is headache and, if the tear ducts are involved, lachrymation. After two or three days the swelling subsides, the secretion becomes thicker and muco-purulent, and the inflammation disappears in a week or two. The constitutional symptoms regularly vanish with the acute condition. The senses of smell and taste are often markedly impaired for some time after an attack.

Complications.—Acute coryza is often only part of a general inflammation of the nose, throat, and bronchi, or the process beginning in the nose extends to the lower parts of the respiratory tract. Otitis media from extension through the naso-pharynx and Eustachian tubes is not uncommon. At times the process extends into the antrum or the frontal sinuses with resulting severe local symptoms.

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Treatment.—Rest in bed is best for all severe cases, and is essential for children and the aged. Free diaphoresis and evacuation of the bowels are desirable at the onset. A hot bath, followed by hot tea or lemonade, with Dover's powder, 10 grains, or 5 to 10 grains of aspirin, will usually cause free sweating. As a laxative two or three grains of calomel may be given at night, with a Seidlitz powder or other saline in the morning. Locally a mild antiseptic spray, such as Dobell's solution, either full strength or diluted to one-half strength, should be used every two or three hours. If there is much obstruction adrenalin chloride 1/2000–1/5000 will usually give relief. Later astringent applications, such as argyrol (20 per cent.) or nitrate of silver, will serve to reduce the inflammation and check the secretion. The influence of internal medication is doubtful, but small doses of Dover's powder, or combinations of camphor, extract of belladonna, and quinine are commonly given.

AUTUMNAL CATARRH
(Hay Fever)

Definition.—An inflammation of the nasal mucous membrane, due to the action of the pollen of certain plants and grasses, and frequently associated with asthma.

Etiology.—The disease appears to have its basis in a hypersensitive condition of the nasal mucous membrane peculiar to certain individuals. This susceptibility is marked in some families and is frequently inherited. The young and middle-aged suffer most often, and the tendency seems to diminish in old age.

The active excitant is regularly the pollen of certain plants and grasses. Dunbar has found that the pollen of many plants and grasses may be active. Different pollens are concerned in the production of the spring and autumnal catarrhs. For the spring hay-fever of the United States the pollen of grasses and sedges seems most effective, though a number of plants also produce active pollen. In the autumn the spring and autumnal catarrhs. For the spring hay fever of the agent. It has been found that the poisonous principle in these pollens is a toxalbumin, which can be readily extracted.

Symptoms.—The disease appears about the first of May or in the autumnal type in August. The onset is marked by sneezing, which is quickly followed by lachrymation and a profuse watery coryza. The nasal mucous membrane becomes swollen and the nares are blocked. There is more or less tickling, itching, or pain in the eyes, nose, and throat. The patients are depressed and languid. Temperature and pulse are normal. Asthma may develop at any time. These symptoms persist for about six weeks and then clear up. The attacks are repeated yearly at the same time.

Treatment.—Attacks may be avoided by going to sea, or by residence in places where the vegetation is scant. Various mountain resorts are popular. Dunbar has perfected a serum from which he claims good results. The serum must be given daily throughout the hay-fever season.

As a palliative a nasal spray of adrenalin chloride 1 to 2000 to 1 to 5000 may be serviceable. Many patients have been improved or cured by the removal of hypertrophied turbinates, or nasal spurs, or adequate treatment of other pathological conditions of the nose.

EPISTAXIS

Bleeding from the nose is a symptom rather than a disease in itself. The causes are either local or constitutional. The local include (1) trauma of any kind, picking the nose, violent sneezing or coughing, and the like; (2) the presence of foreign bodies; (3) neoplasms, which are very rare. The general include (1) a tendency present in growing children, especially the delicate and rheumatic; (2) the onset of acute infections, especially typhoid fever; (3) abnormal conditions of the blood, such as hemophilia, purpura, scurvy, pernicious anemia, or leukemia; (4) chronic nephritis and cirrhosis of the liver; (5) vicarious menstruation; (6) the changes of pressure induced by ascending mountains.

Symptoms.—The blood flows from one or both nostrils. If the patient is lying down the blood may be swallowed and later vomited, or it may be coughed up. In most cases there are no other symptoms. If the loss of blood is excessive, the patient becomes weak and syncope may occur. Death from nasal hemorrhage is very rare except in cases of hemophilia.

Treatment.—In most cases the hemorrhage ceases spontaneously. The application of ice to the nose or snuffing up ice-water will hasten the cessation. The head should be held up. Plugging the anterior nares with absorbent cotton is usually all that is necessary. In severer cases a spray of adrenalin chloride 1 to 2000 to 1 to 5000 may suffice. If not, both anterior and posterior nares should be firmly packed with gauze. Packing the posterior nares may be done by means of a catheter through the tip of which a silk thread is passed into the pharynx and thence drawn out of the mouth for the attachment of the post-nasal pad of gauze. Belocq's canula, if available, renders this procedure much easier. Such packing may be kept in place for not more than 48 hours. The danger of infection in these cases is great, and asepsis should be practised as far as possible. Calcium lactate may be given, 20 to 30 grains thrice daily, and injections of serum, as for hemophilia (see page 257), resorted to if other measures fail.

DISEASES OF THE LARYNX

ACUTE LARYNGITIS

This affection is frequently part of a general catarrh of the respiratory tract. It may occur alone, either as an independent or as a secondary process.

Etiology.—It is caused by exposure to cold, the inhalation of irritating gases, the swallowing of corrosive liquids, or excessive speak-

ing. It occurs in measles and influenza and other acute infectious diseases as part of the catarrhal inflammation of the respiratory tract. Acute rhinitis or pharyngitis frequently involves the larynx by extension.

Symptoms.—Discomfort on swallowing, hoarseness, a croupy cough, and rarely aphonia. The temperature is usually but little raised and the constitutional symptoms are slight. In children acute laryngitis is often associated with severe dyspnea, as in spasmodic croup. In adults edema of the larynx may occur, but is rare. Laryngeal examination shows congestion and swelling of the vocal cords and the adjacent parts of the larynx.

Course.—The affection regularly subsides in a few days or at most a week. An acute bronchitis frequently follows from the downward extension of the process.

Treatment.—Rest for the larynx is important. The milder cases often require no other treatment. Various simple lozenges may be given for the patient's comfort. Children and adults with severe symptoms should be put to bed, and the air of the room kept warm and moist. Inhalations of steam impregnated with the compound tincture of benzoin (℥i to the pint of boiling water) should be given for fifteen minutes three or four times a day. Cold applications or an ice-bag to the throat may help. Children with dyspnea should be treated as for spasmodic croup.

CHRONIC LARYNGITIS

Etiology.—Repeated attacks of acute laryngitis, persistent over-use of the voice, the constant inhalation of dust or tobacco smoke, may be the cause. Nasal obstruction often plays an important part.

Symptoms.—The voice is hoarse, and there is a hoarse cough. Pain or constitutional disturbances are usually lacking. On examination the vocal cords are slightly congested, lustreless, and possibly thickened, and there is some swelling and congestion of the aryepiglottic folds.

Diagnosis.—Care must be taken to exclude other forms of chronic laryngitis, especially the tuberculous and syphilitic. (See pp. 5 and 6.)

Treatment.—The cause, if known, must be removed. Nasal obstruction, if present, must be relieved. For the laryngitis itself, rest and astringent applications either by brush or spray of 3 per cent. nitrate of silver, 3 per cent. perchloride of zinc, or 10 per cent. argyrol may be employed. The general condition of the patient must be improved by all possible means. A mild, equable climate with an atmosphere free from dust is favorable.

EDEMATOUS LARYNGITIS
(Edema of the Glottis)

Etiology.—This very serious affection is met with as a complication (1) of either acute or chronic laryngitis; (2) of severe inflammation of the throat, such as diphtheria, or of the neck, such as erysipelas or

cellulitis; (3) of acute infectious diseases, such as typhoid, typhus or scarlet fever; (4) acute or chronic Bright's disease; (5) angioneurotic edema.

Symptoms.—The picture is that of sudden, rapid strangulation, with rapidly increasing dyspnea, hoarseness, stridor, cyanosis, and, unless relief is secured, death from cardiac failure. On laryngoscopic examination an enormous boggy swelling of the epiglottis, the aryepiglottic folds and adjacent parts may be seen. In some cases the swelling of the epiglottis may be seen on direct inspection and may be felt by the finger.

Treatment.—Cracked ice should be given by mouth and also applied externally; spraying the throat with cocaine, 4 per cent. or better, adrenalin chloride 1 to 2000, may give relief. If not, the edematous parts may be scarified. Finally intubation or tracheotomy may be required.

TUBERCULOUS LARYNGITIS

Etiology.—The disease is rarely primary in the larynx. Almost invariably examination of the lungs will reveal a focus at one or both apices.

Morbid Anatomy.—Beginning as an anemia or hyperemia, the disease soon shows its characteristic tubercles in and upon the epiglottis, aryepiglottic folds, and the interarytenoid tissues. The tubercles later break down, leaving broad ulcers. The disease may extend and involve the pharynx or esophagus. In some cases the ulceration is deep and the cartilages of the larynx are destroyed.

Symptoms.—(1) The disease begins with slight hoarseness, which gradually increases to complete aphonia. (2) Cough and expectoration develop later. The cough may be paroxysmal and exceedingly troublesome. (3) Dysphagia occurs in the more advanced cases and may be intense. If the epiglottis is destroyed every attempt to swallow produces great pain and choking. (4) The patients regularly show the fever, emaciation, and other symptoms, of pulmonary tuberculosis.

Laryngoscopic examination in the early stages shows only an anemia with thickening due to infiltration of the laryngeal mucous membrane, especially that over the aryepiglottic folds. Later all the structures about the glottis become greatly thickened, and broad, shallow ulcers, with gray bases and ill-defined edges, appear. In the final stages the epiglottis may be destroyed, and the ulcers extend into the pharynx or esophagus. The lungs give the physical signs of pulmonary tuberculosis and the sputum shows the bacilli, but not always on the first examination.

Treatment.—These cases must for the most part be treated as cases of pulmonary tuberculosis. Locally inhalations of compound tincture of benzoin may give relief in the early stages. Later applications of lactic acid in glycerin, 5 per cent. at first, increasing gradually to 50 per cent., are advocated. Intratracheal injections of menthol, 1 per cent.

in olive oil, 1 to 3 drams at each sitting, may be given. For the relief of distress anesthesin may be used as a powder or as a 5 per cent. suspension in oil. In the final stages cocain or morphine may be required.

SYPHILITIC LARYNGITIS

The larynx may be involved in either the secondary or tertiary stage of syphilis.

Symptoms.—Hoarseness first calls attention to the larynx, later the voice may be lost. Some cough and expectoration are present, and in the severer forms dysphagia. Edema of the glottis may occur in severe cases. Examination of the larynx shows in the secondary stage a congestion indistinguishable from simple laryngitis, or superficial ulceration which may be extensive. Mucous patches or condylomata are rare. In the tertiary stage gummata may be seen, especially at the base of the epiglottis, or deep ulcers when the gummata break down. Later extensive cicatrization which may cause stenosis of the larynx results. The laryngeal lesions of inherited syphilis are of like character, gummata, ulcers, and cicatrization.

Diagnosis.—The diagnosis of syphilis can usually be safely made from the history and the associated symptoms in the skin, glands, and throat. The Wassermann reaction should be tried and if ulcers are present the spirochete may be sought.

Treatment.—The constitutional treatment of syphilis must be thoroughly carried out. The tertiary lesions are always refractory to treatment. Tracheotomy may be required.

NEUROSES OF THE LARYNX

Two forms of spasm of the larynx are recognized and described as Laryngismus Stridulus and Spasmodic Croup.

1. **Laryngismus Stridulus.**—This affection is peculiar to infants from six months to two years of age, especially the rachitic. It is characterized by recurrent attacks of spasm of the adductor muscles of the larynx. These attacks are often excited by reproof or punishment, and are spoken of as "passion fits" or attacks of "holding the breath." The affection is thought by some to be closely related to the attacks of dyspnea associated with enlargement of the thymus, the so-called thymic asthma; by others it is considered a manifestation of tetany.

Symptoms.—The attacks occur either by day or night. The child suddenly stops breathing, becomes cyanotic, and looks as though about to die, or in some cases struggles for breath, which, the spasm relaxing, is finally drawn in with a long stridulous cry, the so-called "crow." Convulsions may occur with the attack. The attacks recur frequently in some cases. In rare instances death occurs.

Treatment.—The underlying rachitis must be appropriately treated, by fresh air, good food, and sunshine. Daily cold sponging of the back and chest, while the child sits in a warm bath, is very helpful.

2. **Spasmodic Croup** is an affection of children from two to five

years of age. Often it is part of an acute catarrhal laryngitis, in other cases it appears to be an independent disease. The presence of adenoids or enlarged tonsils predisposes a child to this disease. The child having been a little hoarse in the afternoon, or perhaps perfectly well, falls asleep as usual, to wake during the evening or late at night with a hoarse, croupy cough and some dyspnea. In the milder cases the disturbance is slight and the child quickly falls asleep again. In the severer cases the cough persists, the dyspnea increases, cyanosis develops, with retraction of the suprasternal and infracostal regions, in fact all the symptoms of laryngeal stenosis. Sooner or later the attack subsides, the child falls asleep, and wakes in the morning perfectly well or with only a slight hoarseness. The attack may be repeated on several succeeding nights. Recovery always occurs.

Diagnosis.—It is important to distinguish this affection from diphtheritic laryngitis. This is usually easy from the suddenness of the onset, the promptness of relief under appropriate treatment, and the absence of any diphtheritic lesion of the tonsils or pharynx, yet mistakes are made from time to time with very serious results. A culture should always be made from the throat, as the safest means of differentiation.

Treatment.—For the attack the syrup of ipecac, one-half to one teaspoonful, repeated till vomiting is produced, is usually sufficient. Inhalations of chloroform are also effective. Steam inhalations or a hot mustard foot-bath may serve in mild cases. Enlarged tonsils and adenoids should be removed.

DISEASES OF THE BRONCHI

ACUTE BRONCHITIS

Definition.—An acute catarrhal inflammation of the mucous membrane of the bronchi. It is frequently associated on the one hand with tracheitis, on the other with an inflammation of the finer bronchi, the so-called capillary bronchitis.

Etiology.—Bacteria are the active agents. Many are normally present in the bronchi, such as strepto-, staphylo- and pneumo-cocci, Friedlander's bacillus. Certain conditions favor the action of bacteria: (1) The invasion of acute infectious diseases, especially measles, influenza, whooping-cough, typhoid fever, and tuberculosis. Possibly the specific agents of these diseases are the direct excitants of the bronchitis. (2) Influences lowering the vitality or resistance of the patient: (a) Old age and childhood. (b) Exposure to cold and wet. The winter months are, therefore, the favorite season for bronchitis. (c) Sedentary or indoor life. (d) Various diseases, such as Bright's disease or gout. (3) Influences directly influencing the bronchial mucous membrane: (a) Extension of a coryza or laryngitis. Bronchitis is frequently the sequel of an inflammation beginning in the nose or throat. (b) Inhalation of dust, or irritant vapors, such as bromine or formalin. (c) Adenoids and

enlarged tonsils by causing mouth-breathing lead to frequent attacks of bronchitis.

Pathology.—The affected portions of the mucous membrane are congested, swollen, and covered by an abundant mucous or mucopurulent secretion. Microscopically desquamation of epithelium, edema of the mucosa and submucosa, possibly infiltration with leucocytes may be demonstrated.

Symptoms.—The onset of acute bronchitis may be either gradual or sudden. The characteristic symptoms are cough and expectoration. The cough is usually frequent, harsh and paroxysmal. The expectoration is mucous or mucopurulent, scanty at first, becoming more abundant and more purulent as the bronchitis subsides. Infants and young children do not expectorate, but raise the mucus into the throat and then swallow it. Severe bronchitis may give rise to pain under the sternum, especially after coughing, or in either hypochondrium from straining of the diaphragm and abdominal muscles. In vigorous adults constitutional symptoms are lacking or limited to slight fever and malaise. In children and the aged acute bronchitis may be severe, with fever of 102° to 103°, rapid pulse, some dyspnea, possibly cyanosis, loss of appetite, and marked prostration. The physical signs consist of râles, which may be crepitant, subcrepitant, coarse, or piping, either localized or heard diffusely over both lungs.

Course.—The fever and constitutional symptoms should subside within a week or ten days; the cough and expectoration may then disappear or they may continue for several weeks. The severer cases, especially in children and the aged, may readily develop into bronchopneumonia.

Diagnosis.—The symptoms and physical signs combined make the diagnosis easy. We must remember that an acute bronchitis often ushers in measles, whooping-cough, or typhoid fever. In every protracted case tuberculosis must be excluded by careful observation, examination of the sputum, and, if necessary, tuberculin tests. Influenza may be distinguished only by the severer constitutional symptoms or by the demonstration of Pfeiffer's bacillus in the sputum, although, as stated under influenza, the latter is often lacking in cases classed as influenza. Examination of the sputum in acute bronchitis shows it to be composed of mucus, desquamated epithelium, leucocytes or pus cells, and bacteria. The organisms commonly found are the streptococci, staphylococci, pneumococci, and Friedlander's bacillus.

Treatment.—Mild cases often go untreated or require only a sedative cough mixture to relieve the cough at night. Severer cases may be treated at the onset by a hot bath, hot drinks and Dover's powder, as described under coryza. Rest in bed for several days should then be required. The diet should be light and the bowels kept open by laxatives. Counterirritation by camphorated oil for children or by mustard pastes, one part mustard to three or four parts flour, for adults helps to relieve congestion and allay cough. For scanty expectoration and frequent cough,

inhalations of steam with compound tincture of benzoin are useful. The use of the many cough mixtures is steadily diminishing, because they so frequently nauseate the patient and interfere with feeding, which is of the utmost importance. In the early stages small doses of codein (gr. ¼) or heroin (gr. ¹⁄₁₂–¹⁄₁₆) may be given to adults when necessary to allay the cough and permit sleep. Sedatives of this kind possibly increase the danger of bronchopneumonia and should be given with caution to children or the aged. In the late stages stimulating expectorants such as ammonia, senega or squills may be employed. Change of air to a milder climate will often hasten the cure. Cod liver oil and other tonics are of value in convalescence.

CHRONIC BRONCHITIS

Definition.—A chronic inflammation of the bronchi, usually the larger and medium-sized tubes.

Etiology.—(1) Repeated attacks of acute bronchitis. (2) Chronic congestion due to heart disease, or diseases of the lungs, such as emphysema and tuberculosis. (3) Constitutional diseases, such as gout and chronic nephritis. (4) Mouth-breathing from adenoids or other causes.

Morbid Anatomy.—The walls of the bronchi are thickened by chronic inflammation or thinned by dilatation. The mucous membrane of the bronchi shows desquamation or ulceration. The lesions of the associated diseases are found. Emphysema and bronchiectasis regularly accompany the condition.

Symptoms.—Chronic cough and expectoration are the chief symptoms. (1) The cough is usually worse in the morning, owing to the accumulation of secretions during the night. Once the bronchi have been emptied, relief is had for some time, possibly for the day. During the summer the cough improves, or disappears, only to return with cold weather and renewed attacks of acute bronchitis. During the exacerbations the patient may have fever. (2) The expectoration may be very slight, the so-called dry catarrh, or so abundant as to merit the designation of bronchorrhea. In some cases it becomes putrid. It is regularly most abundant in the morning. (3) Dyspnea develops sooner or later from the accompanying bronchiectasis and emphysema. In the later stages the right heart fails with increasing dyspnea, cyanosis and anasarca.

Course.—The disease is protracted through many years, possibly for the lifetime, with improvement in summer, relapses or exacerbations in winter. Recovery in the early stages is not difficult.

Physical Signs.—In the milder cases there may be no abnormal signs on examination. Usually there are subcrepitant, sibilant or sonorous râles to be heard, especially over the lower and posterior parts of the chest. In advanced cases there may be the signs of bronchiectasis, emphysema and dilatation of the right heart.

Diagnosis.—Tuberculosis must be excluded by repeated examinations of the sputum, possibly by tuberculin tests. Asthma can be recognized by the history of the patient and the paroxysmal dyspnea. Emphysema

gives characteristic physical signs. The possibility of the presence of an aneurism or tumor pressing on the bronchi must be remembered.

Treatment.—A mild and equable climate is advisable. The colder months of the year should be spent in Florida, Southern California, Arizona, or the South of France. Warm clothing and avoidance of exposure are important. Careful attention must be given to the digestion and general health of the patient. For the cough itself many remedies are used. Full warm baths are recommended. A combination of sodium bicarbonate 15 grains, sodium chloride 5 grains, spirit of chloroform 5 minims, in anise water may be given in warm milk or water for the morning cough. Terebene in ten minim doses, creosote in 2 or 3 minims, or terpene hydrate 2 to 5 grains, may be given three or four times a day.

FIBRINOUS BRONCHITIS

Definition.—A rare disease, of either acute or chronic course, characterized by paroxysms of dyspnea and cough in which casts of some of the bronchi are expelled.

Etiology.—The disease may be idiopathic or occur as a complication of various infectious diseases, such as diphtheria, pneumonia, measles, scarlet fever, erysipelas, typhoid, variola, rheumatic fever, or influenza. The diphtheria bacillus, the pneumococcus and various other organisms have been found in the casts.

Morbid Anatomy.—The casts represent moulds of small parts of the bronchial system as a rule; in rare cases the whole bronchial system may be reproduced. They are composed of mucin or fibrin.

Symptoms.—The clinical picture is that of a bronchitis, either acute or chronic, in which from time to time occur paroxysms of dyspnea and cough during which the casts are coughed up. Fever occurs intermittently during the course of the disease. The expulsion of the casts may be followed by free hemorrhage.

Diagnosis.—The casts alone identify the disease. The presence of diphtheria or pneumonia must be excluded.

Prognosis.—In the idiopathic form death is rare, in the secondary type it is not infrequent. There is a marked tendency to recurrence.

Treatment.—The treatment is that of bronchitis, acute or chronic. Inhalations of steam or the volatile oils are recommended.

BRONCHIECTASIS

Definition.—A local or general dilatation of the bronchi.

Etiology.—This condition is never primary, but results from two factors: (1) inflammation of the walls of the bronchi, as in bronchitis or bronchopneumonia, weakening all the tissues of the wall; (2) increased intrapulmonary pressure, such as is caused by cough. The condition, therefore, follows (1) bronchopneumonia, especially in influenza or whooping-cough, or tuberculosis; (2) chronic bronchitis and emphysema; (3) compression of the bronchi by tumors or aneurism; (4) impaction of

a foreign body in a bronchus; (5) interstitial pneumonia and chronic pleurisy. The disease is met with at all periods of life.

Morbid Anatomy.—The dilatation is found localized or distributed generally through one or both lungs. The dilatations are fusiform or saccular. In some cases cavities of large size are produced, the cavities full of mucopurulent, possibly fetid, secretion. Emphysema, some sclerosis of the lung, and possibly chronic pleurisy are frequent accompaniments.

Symptoms.—As a sequel or accompaniment of the conditions named above the patient develops a chronic cough with abundant, foul, or fetid expectoration. The cough is paroxysmal, occurring in the morning or at other times when the dilated bronchi become full. Once they are

Fig. 1.—"Clubbed fingers" in a patient having bronchiectasis. From the collection of Dr. Walter B. James.

emptied the cough ceases for some hours, possibly for the day. Paroxysms of cough and offensive secretion may make the patients wretched both in body and mind. The sputum is thin, mucopurulent in character, often foul in odor, and may amount to a pint or two a day. It contains great numbers of bacteria, degenerated leucocytes and epithelium, crystals of fatty acids, and possibly leucin and tyrosin. The disease lasts indefinitely. Mild cases may recover, the severer go on for years till the patient dies of exhaustion, from bronchopneumonia, or other complication.

The physical signs are those of a localized bronchitis with subcrepitant or large mucous râles, or those of small cavities, at one time full of secretion, giving rise to dulness and diminution or absence of voice and breath sounds, and again empty and giving the usual signs of cavity.

As in many other chronic pulmonary diseases, the terminal phalanges

of the fingers may show enlargement, which by the X-ray can be shown to affect only the soft tissues. (See Fig. 1.)

Diagnosis.—The character of the cough and expectoration usually marks the condition. The physical signs other than the rāles are often difficult to obtain. Tuberculosis must be excluded by the absence of constitutional symptoms, failure to find the bacilli in the sputum, and possibly by tuberculin tests.

Treatment.—The milder cases are treated as cases of chronic bronchitis. Residence in a mild, dry climate is of most value. Intratracheal injections of menthol 10 parts, guaiacol 2 parts, and olive oil 88 parts, are recommended. Single bronchiectatic cavities in accessible positions may be opened and drained surgically.

BRONCHIAL ASTHMA
(Spasmodic Asthma)

Definition.—No satisfactory definition of the affection can be given. The term asthma is often applied to dyspnea of renal or cardiac origin, but should be applied only to the affection here described.

Etiology.—The affection occurs at all ages, most often in males, and especially in persons of neurotic ancestry. Liability to the disease is hereditary in some families. Certain nasal conditions, such as nasal polypi, hypertrophic rhinitis, or adenoids, predispose to it. Hay fever and asthma are closely allied and often associated. In persons subject to asthma the attacks are precipitated by a great variety of causes; the exciting cause is often peculiar to the individual. To one, residence in the city is harmful, to another the country. The odor of certain flowers, or animals, or the pollen of certain plants will cause attacks in some cases and not in others, and so on. Fright or excitement of any kind, exposure to cold or wet, disturbances of digestion and the like, are all given as possible causes of attacks.

THEORIES.—There are several theories of the causation of the dyspnea: (1) Spasm of the muscular coats of the smaller bronchi. (2) A special form of inflammation or swelling of the bronchial mucous membrane, with a peculiar exudation, the bronchiolitis exudativa of Curschmann. (3) Spasm of the diaphragm and possibly of other muscles of respiration. (4) The phenomena excited by injections of foreign serum in animals already sensitized by previous injections sometimes closely resemble asthma. In man paroxysms of severe or even fatal asthma have been precipitated by injections of diphtheria antitoxin (horse serum). This accident has occurred especially in those who have previously suffered from asthma. Meltzer has suggested that asthma is therefore an anaphylactic reaction excited in sensitized persons by inoculation with foreign albumins (pollen, etc.).

Symptoms.—The attacks of dyspnea, which constitute the essential feature in asthma, come suddenly without warning, or may be preceded by disturbances of digestion, nervousness, or depression which enable the

patient to foretell an attack. Nocturnal attacks are frequent. In the milder attacks the patient suffers only slight dyspnea and an annoying cough with tenacious expectoration. In the typical form the dyspnea is extreme, the patient sits up, supporting the shoulders by clasping the arms of a chair or resting the elbows on a table, in order to give more effective play to the accessory muscles of respiration. Inspiration is quick and jerky, all the muscles of respiration, excepting the diaphragm, which is rigid, being thrown into violent action. The muscles of the neck especially stand out. Despite the effort, but little air enters the chest. Expiration is prolonged to several times the normal time. Both inspiration and expiration are accompanied by loud wheezing and piping sounds which may be audible at some distance from the chest. The total number of respirations per minute is little, if at all, increased, and the pulse is but little disturbed. The face is pale, or livid, frequently bathed in cold sweat, the expression anxious, speech difficult or impossible, and in severe cases death seems inevitable. Sooner or later, however, the spasm relaxes, air enters the chest more easily and the attack after lasting two or three hours subsides. Usually some dyspnea remains for several days and the severe symptoms recur at any time. During the critical period there is little or no expectoration. With relaxation the patient coughs and expectorates freely. The sputum is thick, tenacious, consisting chiefly of mucous plugs or masses. Floated in water, these masses may show the form of Curschmann's spirals. Microscopically the sputum shows numerous eosinophile cells and possibly the Charcot-Leyden crystals. The spirals consist of mucin; the composition of the crystals is unknown. The blood shows a leukocytosis and an eosinophilia. The paroxysms of asthma may be repeated for days or weeks. Usually after a paroxysm the patient is free for weeks or months, till an attack is brought on again by some of the exciting influences. In chronic cases the patients regularly develop emphysema and chronic bronchitis.

Prognosis.—The asthmatic attack is practically never fatal and yet the disease recurs through life. In some the attacks tend to increase in frequency, in others with care they can be made less frequent.

Treatment.—For the paroxysm a hypodermic injection of morphine, $\frac{1}{6}$ to $\frac{1}{4}$ grain, usually gives relief. Atropine may be given with it. Of recent years remarkable success has been obtained from the hypodermic use of adrenalin chloride, 10 minims of the 1/1000 solution being given. The fumes of stramonium leaves, in powder or cigarettes, may give relief. Nitre paper is similarly used. In the intervals careful examination of the nose and throat must be made and adenoids or enlarged tonsils, nasal polypi or hypertrophied turbinates removed. The general health must be improved by attention to diet and manner of life. Change of climate may relieve when other means fail. Each individual must select a place for himself, where he is free from attacks. The administration of iodide of potassium or arsenic may be tried. In some persons aspirin in doses of 10 grains two or three times a day will prevent attacks or afford relief during them.

BRONCHOPULMONARY HEMORRHAGE

Hemorrhage from the lungs, or hemoptysis, occurs from a great variety of causes.

(1) Tuberculosis of the lungs, from the rupture of large or small vessels. Hemorrhage may be the initial symptom, or may occur at any time during the disease, and in chronic tuberculosis is not rarely the final event. (2) Other inflammatory lesions of the lungs, such as pneumonia, abscess, or gangrene, or even bronchiectasis or emphysema. (3) Congestion of the lungs from heart disease, especially lesions of the mitral valve, or from infarctions resulting from endocarditis. (4) Arteriosclerosis affecting the pulmonary vessels, especially in the gouty. (5) New growths of the lung. (6) Trauma from blows upon the chest or falls, especially from puncture by broken ribs. (7) The strain of heavy lifting. (8) Aneurisms of the aorta or pulmonary vessels rupturing into the lung. Erosions in the bronchi may be caused by pressure of aneurisms and bleeding follow without actual rupture. (9) Pulmonary diastomatosis, invasion of the lung by parasitic flukes in Japan and China sometimes gives rise to hemorrhage from the lungs. (10) Ulcerative lesions of the larynx, trachea, or bronchi. (11) Hemoptysis may occur in the hysterical and neurotic without discoverable cause. (12) Scurvy, leukemia, purpura, and the malignant types of acute infectious diseases, are sometimes complicated by hemoptysis. (13) Vicarious menstruation is a rare cause. (14) Finally pulmonary hemorrhage not infrequently occurs in young persons without discoverable cause and these individuals may never develop tuberculosis or any other disease to explain the occurrence.

Symptoms.—The blood usually appears suddenly and unexpectedly. The patient feels a tickling in the throat, coughs, and expectorates blood. The amount brought up varies from a trace to a pint or more. Usually the actual amount of blood lost is small, possibly an ounce or two. The associated symptoms vary greatly. The patient may not be disturbed at all. Frequently they are greatly excited and anxious, the face flushed, the pulse rapid and bounding. Only in rare cases do they lose sufficient blood to give the constitutional symptoms of hemorrhage, pallor, breathlessness, sweating, rapid, small and weak pulse, and corresponding depression.

The physical signs vary with the cause. During the attack of hemorrhage the usual examination of the chest should be omitted altogether, or limited to auscultation of the natural breathing.

Diagnosis.—The chief point is to make sure that the blood comes from the lungs. Blood flowing from the nose may run into the throat and then be coughed up. The nose, throat, and larynx should be carefully examined. The stomach is the only other probable source. Blood from the lungs is bright red, frothy, alkaline. That from the stomach is regularly altered in some degree, mixed with food or mucus, probably

acid from such admixture, and often "coffee-grounds" in appearance. Search for the cause of the hemorrhage must often be postponed till all bleeding has ceased. The history of the patient, the conditions under which the hemorrhage occurred, and the results of physical examination will usually lead one to a correct conclusion.

Prognosis.—Only in aneurism or in advanced tuberculosis is pulmonary hemorrhage likely to be fatal. The ultimate outcome depends upon the cause of the bleeding. In certain cases of pulmonary congestion due to heart disease hemorrhage from the lungs may give relief.

Treatment.—Quiet of body and mind is essential. The patient should be put to bed and a hypodermic injection of morphine sulphate, ¼ grain, given. Assurance as to the outcome of the hemorrhage is most valuable. If blood pressure be high, nitroglycerine may be given hypodermatically, or amyl nitrite inhaled. No other treatment is ordinarily called for. Rest should be continued till the bleeding has entirely ceased.

DISEASES OF THE LUNGS

DISTURBANCES OF CIRCULATION OF THE LUNGS

Congestion of the lungs may be either active or passive.

1. ACTIVE CONGESTION occurs (1) in the initial stage of pneumonia, bronchitis, pleurisy, or tuberculosis; (2) from the inhalation of excessively hot or cold air or irritating gases; (3) from violent exertion. The symptoms and physical signs are those of the initial stage of pneumonia, and the treatment the same.

2. PASSIVE CONGESTION.—Two forms are recognized: (a) Brown induration, the congestion of the lung brought about by disease of the heart, chronic endocarditis, myocarditis, or dilatation, which prevents the normal flow of blood from the lungs, or by pressure of aneurisms or tumors upon the pulmonary veins. The capillaries of the lung become distended, some diapedesis and hemolysis take place and chronic bronchitis ensues. Cough and expectoration result and in the sputum leucocytes and epithelial cells containing blood pigment can be found.

(b) HYPOSTATIC CONGESTION is brought about partly by weakness of the heart action, partly by gravity, and is met with in all enfeebled states of the body and especially in the acute fevers, such as typhoid. It is marked in conditions associated with stupor or coma, such as brain lesions, or morphine poisoning. The posterior portions of the lungs are deep, bluish-brown in color, heavy, and dark blood flows freely on incision. Microscopically the veins and capillaries are engorged with blood, and blood is often found in many of the air-vesicles. Physical examination shows deficient pulmonary resonance over the bases of both lungs, with diminished voice and breathing sounds and many coarse, sometimes bubbling, râles.

Treatment.—The condition should be prevented, if possible, by frequently turning any recumbent patient from one side to the other or upon the face, to prevent the settling of the blood in the posterior and

lower parts. The heart action must be maintained by appropriate treatment. Cupping of the chest may be resorted to in extreme cases.

EDEMA OF THE LUNGS.—Exudation of serum into the lungs is rarely primary, but is a feature of the terminal stages of many diseases, especially those of the lungs, the heart, arteries, and kidneys. It occurs especially in patients who already have marked pulmonary congestion with edema in other parts of the body. Not infrequently it follows too rapid removal of pleural effusions. One in every four or five deaths in cases of protracted illness is ushered in by pulmonary edema. The lungs postmortem are very heavy, sodden, and usually congested posteriorly. On section a watery serum exudes freely from the cut surface, either clear or blood-tinged. The cause of pulmonary edema is found in increased blood pressure in the capillaries of the lungs, aided by degenerative changes in the capillary epithelium, possibly bacterial in origin.

Symptoms.—Pulmonary edema usually comes on suddenly with urgent dyspnea, cough and expectoration of frothy watery fluid, if the patient is strong enough to cough and expectorate, cyanosis, rapid, irregular and feeble heart action. Unless promptly relieved death is imminent.

PHYSICAL SIGNS.—The condition is regularly associated with hypostatic congestion, the signs of which are present, and in addition abundant loud, bubbling, liquid râles, which can be readily felt by the hand on the chest wall, as well as heard. These râles may be heard only at the bases or may be present over all parts of the lungs. The mouth and throat may be filled with bubbling watery mucus, and every respiration accompanied by bubbling, the traditional "death rattle" of the novelist.

Treatment.—Dry cupping of the chest, counter-irritation by mustard paste applied to the chest, and rapid cardiac stimulants, such as camphor, strychnine, caffeine, and alcohol, are indicated. Adrenalin chloride, ten minims of the 1/1000 solution, has also been found effective. Artificial respiration may aid. Venesection may be employed if cyanosis and enlargement of the right heart are present.

EMBOLISM, THROMBOSIS, AND INFARCTION OF THE LUNG.—Emboli from various parts of the body may lodge in the lung. The right heart, especially the auricular appendix, is the most frequent source, but they may come from injury or disease of the bones (fat embolism), or from phlebitis such as occurs in the uterine or femoral veins.

Morbid Anatomy.—A branch of the pulmonary artery having been plugged, if the patient lives, the territory supplied by the artery becomes an area of lowered blood pressure and blood from all the adjacent areas settles in it till it becomes intensely engorged and even infiltrated with blood, producing the infarction. Later the blood is absorbed and the infarction becomes an area of fibrosis or scar tissue. If large branches of the pulmonary artery are plugged, the patient dies so promptly that nothing more than the thrombus or embolus is to be found.

Infarcts appear as wedge-shaped or globular areas, firm, deep-red or blackish-red in color, oozing blood on section. Microscopically blood is found in the air-vesicles and infiltrating the interstitial tissue. Finally

only the areas of scar tissue are left. In cases of fat embolism there may be great numbers of plugs of fat found in the finer branches of the pulmonary artery.

Etiology.—Pulmonary embolism is a frequent complication of endocarditis, especially the malignant type. It may follow metritis, or typhoid fever, or injury (fracture) or disease involving the medulla of bones. It is one of the causes of sudden death following operations upon the appendix, uterus, or other abdominal organs.

Symptoms.—With embolism of a large vessel, death follows almost at once with symptoms of sudden dyspnea, cyanosis, and great anxiety. Smaller emboli give rise to some pain in the side, dyspnea, cough, and expectoration of blood. The symptoms may be of brief duration or continue for several days, depending on the size of the obstructed vessel.

PHYSICAL SIGNS.—In the quickly fatal cases no local signs are obtainable. In other cases the infarction may be represented by an area of dulness, at the base of a lung or in the axilla, with diminished voice and breathing; or, if the area be large, bronchial voice and breathing, and fine, subcrepitant or coarse râles.

Diagnosis.—Pneumonia gives similar signs, but the absence of the constitutional symptoms usually renders the diagnosis easy. The sequence of events also usually points directly to embolism.

Treatment is purely symptomatic, for relief of pain and distress.

ATELECTASIS
(Pulmonary Collapse and Compression)

ATELECTASIS, or failure of expansion in the lungs of the new born, is frequently seen in infants who have been asphyxiated during birth or are enfeebled by prematurity or other cause. If the atelectasis is extensive, the infants are cyanotic and breathe feebly, the cry is weak, the pulse is rapid, and they are liable to attacks in which the cyanosis becomes extreme, and death may ensue.

COLLAPSE of the lungs is a similar condition, occurring in children or adults, as the result of obstruction of the finer bronchi by secretions accumulating in them from bronchitis or bronchopneumonia or by pressure upon them caused by tumors or pleural effusions. The extreme grades of the condition such as are seen when an entire lobe or lung is compressed by a pleural effusion have been spoken of as carnification.

In atelectasis and collapse the non-aërated portions of the lungs appear as depressed areas on the pleural surface, deep red or bluish in color from congestion, extending a varying distance into the substance of the lung. With a blow-pipe such areas can be readily inflated and made to appear normal.

PHYSICAL SIGNS.—In infants atelectasis rarely gives appreciable pulmonary signs. Theoretically there should be slight dulness over the lungs posteriorly, but the change is too slight to be of importance. Collapse of the lung in older children is usually obscured by the signs of bronchitis or bronchopneumonia. It may serve to lessen pulmonary resonance and make

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the breathing and voice signs faint. The compressed lung, if the area is large, gives dulness and diminished or absent voice and breath sounds. It is usually the chief factor in the continuance of the signs after removal of fluid.

Treatment.—Atelectasis in new-born infants must be treated by maintaining the body heat, and from time to time stimulating respiration by sprinkling alternately hot and cold water upon the chest, or by spanking. Direct inflation of the lungs has not been successful, and medication is of little value.

In children and adults collapse of the lung is to be prevented, if possible, by the adequate treatment of bronchitis or bronchopneumonia, or the removal of effusions. In bronchitis or bronchopneumonia measures directed to the removal of secretions from the bronchi are of value. Frequent changes of position, especially turning on the face, or lifting the foot of the bed, help drainage. Stimulating expectorants, such as ammonium carbonate or chloride, may be of value. Vomiting may be produced in patients not too debilitated.

In cases where the lung has been compressed by effusions, after the removal of the fluid or drainage of the chest, expansion of the lung may be favored by respiratory exercises. Forced inspiration may be practiced at intervals during the day, either normally or with compression of the sound side. Wolff's bottles, in which water is blown from one to the other, may be used.

LOBAR PNEUMONIA
(Croupous or Fibrinous Pneumonia. Lung Fever)

Definition.—Primary lobar pneumonia is a specific infectious disease marked by a local inflammatory lesion in the lung, a general toxæmia and a sharply self-limited febrile course.

Etiology.—PREDISPOSING CAUSES.—Pneumonia is among the commonest of acute diseases and is widely spread throughout the world, although most frequently encountered in temperate climates. It occurs chiefly during the cold, wet months of the year and is most prevalent in the late winter and spring. No age is exempt, but the disease is commonest in children of between two and six years, in adults of between twenty and forty years, and in the aged. Males are more frequently attacked than females. The sudden lowering of vitality by *exposure to cold* is an important predisposing cause. Malnutrition, alcoholism, overcrowding and chronic diseases also increase the liability to pneumonia. The disease sometimes follows directly upon contusions and other injuries of the chest wall. One attack so far from conferring immunity seems distinctly to predispose to further attacks and two or more repetitions of the disease are by no means rare.

THE EXCITING CAUSE.—Lobar pneumonia, in the vast majority of cases at least, is caused by a specific germ known variously as the *diplococcus pneumoniæ, micrococcus lanceolatus* and *pneumococcus*. Its rela-

tion to the disease was made clear by the work of Fraenkel in 1884 and later by Weichselbaum.

The DIPLOCOCCUS PNEUMONIÆ is an oval or lance-shaped coccus occurring in pairs, with each pair usually surrounded by a faint capsule. The organism is found abundantly in the sputum of pneumonia patients, in the bronchi, in the inflamed areas of the lungs, in the complicating lesions of pleura, endocardium, pericardium, joints, etc. Moreover, it has been demonstrated recently that the germ is present in the circulating blood in most if not all cases during the active stage of the disease. The pneumococcus is also found not infrequently in the mouths of healthy individuals, is capable of producing many other inflammations than those of the lungs and may even cause a general septicæmia without evident local lesion. The harmful effects of the organism seem to be due chiefly to the production of an active toxin—the so-called pneumotoxin—which is present in the blood during the active stage of pneumonia. The germ probably reaches the lung through the respiratory passages, but something more than its mere presence there (e.g., lower resistance of the tissues or increased virulence of the germ) is needed to produce the disease.

It is true that other micro-organisms than the pneumococcus (e.g., Friedländer's bacillus, B. influenzæ, B. typhosus, etc.) seem capable of exciting similar inflammations of the lungs, but there can be no doubt that most, if not all cases, of primary lobar pneumonia are due to infection by the diplococcus pneumoniæ.

Although usually the disease seems but slightly transmissible, its contagious nature is sometimes clearly evident and many small, and a few large, epidemics have been recorded.

Morbid Anatomy.—Usually the greater part, or the whole, of one lobe is involved in the inflammatory process. Not infrequently, however, two lobes of the same lung are affected, and occasionally one or more lobes of both lungs. The lower lobes are much more commonly attacked than the upper, and the right lung rather more frequently than the left. The inflammation is characterized especially by the preponderance of fibrin in the exudate. The process is divided into *four* stages.

1. *Stage of Engorgement.*—The affected part of the lung is dark red, congested and somewhat firmer and heavier than normal, but still contains some air and floats in water. Its cut surface exudes blood. Microscopically the capillaries are seen to be greatly distended with blood. The air vesicles are small and contain only swollen alveolar cells and a few red cells.

2. *Stage of Red Hepatization.*—The lung is now solid, very heavy and of a dull red color. It contains no air and sinks in water. The pleural surface is covered with fibrinous exudate; the cut surface is dry, granular, of brick red color. Microscopically the alveoli are seen to be distended with a thick mesh-work of fibrin enclosing red cells, desquamated epithelial cells, leukocytes and pneumococci. The infundibula and many of the finer bronchioles also are filled with exudate.

3. *Stage of Gray Hepatization.*—The inflamed area is still solid and unaërated but has now a grayish or yellowish gray color and is extremely friable. The cut surface is no longer dry and granular, but exudes a thick, turbid fluid. The microscope shows the alveoli still distended and the capillaries collapsed, but the exudate is no longer chiefly made up of fibrin net-work and red cells. These have, for the most part, disappeared and the exudate consists chiefly of polymorphonuclear leukocytes, granular detritus and serum.

4. *Stage of Resolution.*—The lung becomes less and less solid, the alveoli again contain air, the disintegration, liquefaction and absorption of the exudate continue until the lung tissue is completely restored to integrity. In the disappearance of the exudate expectoration plays no appreciable part. The removal is brought about by absorption and by the action of the phagocytic leukocytes.

The different stages described are by no means always sharply defined, and it is not uncommon to have red and gray hepatization at the same time in different parts of the same lobe.

Symptoms.—The INCUBATION PERIOD is not known, but is usually not longer than a day or two.

The ONSET is regularly abrupt, a severe *chill* being usually the first symptom. Occasionally cough, fever or pain in the chest may precede the chill by a few hours. Occasionally, too, a distinct rigor may be lacking. The temperature rises within a few hours to 103° or 104° F. and remains continuously high. There are the usual headache, prostration and general pains of a severe acute infection. *Pain* in the chest and a short, dry *cough* are early and, usually, distressing symptoms. The patient is apt to lie on the affected side, and with his flushed cheeks, bright eyes, anxious expression and rapid breathing presents a very characteristic and distinct picture. The *fever* rises abruptly with the chill and within 12 hours usually reaches 103°–105° F. This height is thereafter maintained throughout the disease with little variation; the fever chart showing a striking contrast to the regular daily fluctuations seen in typhoid. Some time between the fifth and the ninth day of the disease, most commonly on the seventh day, the temperature suddenly drops within a few hours to normal or thereabouts. This CRISIS is one of the striking features of pneumonia and is usually accompanied by a profuse sweat and by great amelioration of all the symptoms. Not all cases show this sudden and violent drop. Often the critical fall occupies 24 or 36 hours and sometimes, especially in children, the descent is more gradual still—by lysis. Rarely the crisis is seen as early as the third day or is delayed until the eleventh or twelfth day. Occasionally a sharp but temporary drop in temperature (pseudo-crisis) occurs a day or two before the true crisis. Among the aged the fever is apt to be low. (See Figs. 2 and 3.)

The COUGH is a prominent and often a distressing symptom. It is at first short, hard, painful and unproductive. Soon, however, it is accompanied by scanty, tenacious, blood-stained sputum. The blood may at first appear in bright streaks, but it usually shows as a brick red or

orange tinge, and these small, tough, rust-colored sputa are among the important diagnostic signs of the disease. In the later stages the expectoration becomes more abundant, mucopurulent, and faintly yellow or green in color.

PAIN begins usually within a few hours of the onset, is referred as a rule to the axilla of the affected side, is sharp and stabbing in character and is made much worse by a deep breath or by coughing. It is apt to subside in the latter part of the disease, but it may remain intense and distressing throughout. Occasionally it is lacking altogether. Rarely the pain may be referred chiefly to the abdomen.

DYSPNEA.—The breathing rate is increased from the beginning. At first the increase is only moderate (30–40 per minute); later on in the disease, however, especially if the case be doing badly, the rate may reach
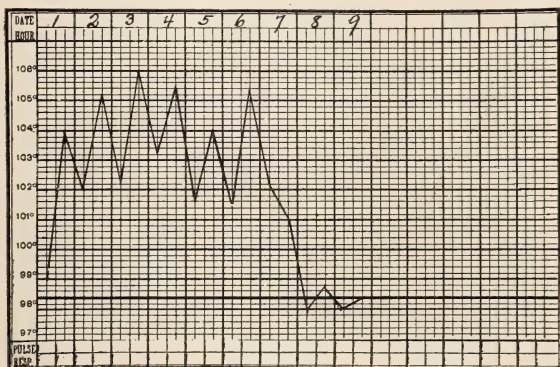
FIG. 2.—Fever of lobar pneumonia. The remissions of the fever are somewhat more marked than is usual.

50 or 60 or even more. At such times the breathing is labored and is attended with cyanosis. In children and some adults expiration is commonly accompanied by an audible grunt.

The PULSE at first is full, bounding, of low tension and moderately increased in rate (100–110). As the disease progresses the rate somewhat increases and the size and force of the pulse lessen. In grave cases the rate may reach 140 or 150. The heart action shows corresponding variations. The sounds at first are loud and clear, but grow progressively fainter as the heart weakens. The second pulmonic tone is usually accentuated and a progressive weakening of this sound is a warning of value as indicating failing strength in the right ventricle. In fatal cases the heart grows progressively weaker and more rapid; the lips, nails and skin generally take on a bluish tinge; pulmonary edema develops; the skin is bathed in cold sweat; cyanosis becomes more and more marked and the patient gradually sinks into stupor and dies.

The BLOOD shows regularly a leukocytosis and an increase in the fibrin constituents. The leukocytes vary usually between 12,000 and 30,000

per c.mm., but may reach 60,000.   The increase affects especially the polymorphonuclear cells, which may form 90–95 per cent. of the total. The absence of a leukocytosis is usually an unfavorable symptom.

NERVOUS SYMPTOMS.—In children a convulsion may replace the initial chill.   Delirium occurs under several conditions.   It is common in the pneumonias of children, in adults with very high fever, in cases complicated by meningitis and especially in alcoholic subjects.   It is usually of the noisy, violent type.   In debilitated subjects and in the aged, however, the delirium is often quiet and muttering.

DIGESTIVE SYMPTOMS are as a rule not prominent.   The tongue is usually heavily coated and moist, but in many of the severe cases becomes dry, brown and cracked.   Vomiting is not uncommon at the onset and may persist.   Constipation is the rule, but diarrhea is not very infre-
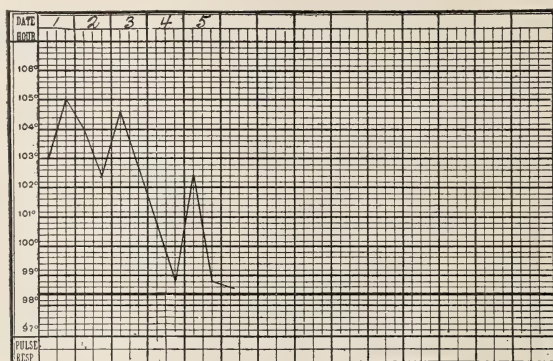
FIG. 3.—Lobar pneumonia with a pseudo-crisis on the third day, a rise of fever again on the fifth day, with a final fall.

quent.   An occasional and very grave symptom is the appearance of marked tympanitic distention of the abdomen.   Jaundice, usually of the non-obstructive type, is sometimes seen.

The URINE during the active stage of the disease is concentrated, highly colored, usually contains a small quantity of albumin and is deficient or altogether lacking in chlorides.

An eruption of *herpes* around the lips and nose occurs in about one-half of the cases.

Physical Signs.—These differ so much at different periods of the disease that it is convenient to describe them separately in the stages of engorgement, hepatization and resolution.

In the FIRST STAGE (engorgement) INSPECTION shows the flushed anxious face, the rapid breathing, the movement of the alæ of the nose and, usually, slight restriction of movement of the affected side of the chest.   By PALPATION this limitation of movement is even better appreciated.   Vocal fremitus is usually not distinctly changed from the normal, but may be slightly increased.   A pleural friction rub is sometimes felt.

PERCUSSION reveals some impairment of normal resonance when compared with the other side. This slight dulness is often accompanied by a distinctly tympanitic quality—the so-called Skodaic resonance. By AUSCULTATION one can detect over the affected area usually a distinct enfeeblement or actual suppression of the normal vesicular murmur. Sometimes, however, the breath sounds are harsh and approach the bronchial type (broncho-vesicular breathing). The transmitted voice is often louder and clearer than normal, but is not distinctly bronchial. A sign of much importance is the *crepitant râle* which is heard regularly during this first stage. It consists of a group of very fine, dry, crackling sounds at the end of inspiration. This stage usually lasts one or two days.

STAGE OF HEPATIZATION.—INSPECTION AND PALPATION show much restriction of movement over the affected portion and exaggerated movement of the other side. Vocal fremitus is distinctly increased. On PERCUSSION there are dulness, amounting often almost to flatness, and marked resistance to the examining finger. If the apex is involved the note has often a tympanitic quality as well.

AUSCULTATION reveals complete disappearance of the normal vesicular murmur and its replacement by a loud, high pitched bronchial sound both on inspiration and expiration. The voice is greatly intensified and clear (bronchophony), as is also the whisper. Râles are usually lacking in this stage.

STAGE OF RESOLUTION.—With the beginning of resolution there is a gradual return of all the signs to normal. The chest moves more freely and the percussion note becomes less and less dull (although it may be some time before it is perfectly resonant). The bronchial breathing grows softer and lower pitched, is audible only with expiration and finally is replaced by the vesicular murmur. The râles return in this stage and are usually very abundant. Some are of the crepitant type, but most of them are moist râles of different degrees of coarseness. They are heard chiefly with inspiration.

Variations in Physical Signs.—The signs of the first stage may last but a few hours or may be protracted for several days. Indeed, dulness and bronchial breathing may be lacking altogether, if the consolidation is small and is deep in the lung. Bronchial breathing is absent also in cases where the bronchi are occluded by mucus or by fibrinous exudate (massive pneumonia).

The signs of resolution usually appear about the time of the fall in temperature, but sometimes resolution is almost completed before the crisis occurs. On the other hand, resolution may be much *delayed* and dulness and bronchial breathing may persist for some days or even weeks after the temperature has fallen. A complicating pleural affection may greatly modify the physical signs and lead to much confusion.

SPECIAL FORMS.—The term DOUBLE pneumonia is applied to cases in which both lungs are involved. If the consolidation is small and deep-seated (CENTRAL pneumonia) the physical signs are apt to be slight and indistinct and very slow in developing. Instances of MASSIVE pneumonia

are seen rarely in which not alone the lung parenchyma but the bronchi as well are filled with fibrinous exudate. The transmission of the bronchial breath and voice sounds is thus prevented and these important signs may be altogether lacking.

CREEPING or MIGRATORY pneumonia is that form in which the inflammatory process attacks various portions of the lung in succession.

When, after the usual onset, the disease subsides in the course of two or three days without going on to its full development it is spoken of as LARVAL pneumonia. An important form is that known as ASTHENIC or TOXIC pneumonia in which the symptoms of toxemia are marked and out of all proportion to the extent of the local process. This type is common in aged and debilitated subjects and in drunkards.

The term CEREBRAL pneumonia is sometimes applied to cases in which the nervous symptoms are conspicuous. Such cases may closely simulate meningitis.

SECONDARY pneumonias occur as a complication of such diseases as typhoid fever, influenza, diphtheria, plague, etc. They differ from primary lobar pneumonia in their etiology, their histological features and their clinical course. The same may be said of *terminal* pneumonias which often develop in the last days of chronic exhausting diseases, which give only slight and indistinct symptoms and which are very apt to be overlooked.

COMPLICATIONS.—In view of the active pathogenic qualities of the pneumococci and of the fact that in most if not all cases of pneumonia they are carried to all parts of the body in the circulating blood, it seems somewhat strange that serious complications are not more frequent even than they are.

The Lung.—Instead of proceeding to resolution in the usual way a part of the hepatized area will occasionally suppurate, break down and form an ABSCESS, or the blood supply of a certain portion may be cut off and GANGRENE result. On the other hand, the consolidated area instead of resolving or breaking down may be the seat of a productive inflammation with development of new connective tissue, organization of the exudate and permanent damage from FIBROID INDURATION or CIRRHOSIS. These complications are not common. DELAYED RESOLUTION has already been spoken of. Contrary to popular belief, tuberculosis is rarely, if ever, engrafted upon a lobar pneumonia. Such apparent instances are from the outset really cases of tuberculosis of the acute pneumonic type simulating lobar pneumonia.

The Pleura.—Affections of the pleura are among the most frequent and important of complications. A fibrinous inflammation of the pleura covering the inflamed lobe is rarely lacking and is hardly to be classed as a complication. PLEURISY WITH EFFUSION develops frequently in the latter part of the disease and, usually, so insidiously that frequent and careful physical examinations may be needed to demonstrate its presence. EMPYEMA, though less frequent than serous effusion, is by no means uncommon. The pus usually contains pneumococci in large numbers.

The Circulatory System.—Next to serous pleurisy and empyema PERI-CARDITIS is probably the most frequent of the serious complications. It is especially apt to develop in double and in left-sided pneumonia and is said to be commoner in children than in adults. It may be fibrinous, serous or purulent in character. It is sometimes difficult of recognition even when repeatedly and carefully sought for.

ACUTE ENDOCARDITIS is also not uncommon. Usually it is of the malignant or ulcerative type. It attacks both the right and left side of the heart and in the septic thrombi upon the affected valves the *diplococcus pneumoniæ* is usually to be found in pure culture.

Venous thrombosis and embolism of the cerebral or other arteries are rare complications.

MENINGITIS is a grave and not very infrequent complication. It is due to infection of the meninges by the pneumococcus. *Acute arthritis, acute nephritis, jaundice, croupous colitis* or dysentery, *parotitis, otitis media* and *neuritis* are occasionally met with.

Diagnosis.—The disease is usually easily and promptly recognized. The chill and rapid rise of temperature, the sharp pain, the cough and rusty sputa, the rapid breathing and the characteristic physical signs furnish, in typical cases, an unmistakable clinical picture. Even when the physical signs are late in developing the other symptoms are usually sufficient for a prompt diagnosis. In children and in alcoholic subjects the cerebral symptoms may be so pronounced as to mask the true nature of the trouble. In debilitated and aged patients, too, the onset may be insidious and the rational symptoms slight and indefinite. A careful physical examination, however, will usually overcome the difficulty in all these cases. In rare instances the pain may be referred to the abdomen and may be associated with muscular rigidity. Occasionally such cases have been mistaken for appendicitis or gall-bladder disease. Pleurisy with effusion and empyema, especially in children, sometimes give physical signs which closely simulate those of consolidation. There may be loud bronchial breathing and bronchophony and even little or no diminution in vocal fremitus. In such cases the exploratory needle may be needed to clear up the diagnosis.

The Blood.—The leukocyte count is regularly increased, often to 30,000 and sometimes to more than 50,000. Such a leukocytosis, coupled with the rational symptoms of pneumonia, makes the diagnosis highly probable even in the absence of definite physical signs.

An equal increase is rarely met with except in cases of active suppuration, or cerebrospinal meningitis. The percentage of polynuclears is usually increased.

Pneumonia may, however, be accompanied by a normal leukocyte count or even a leukopenia. With severe constitutional symptoms a leukopenia is of unfavorable import.

Blood cultures may yield the pneumococcus, but usually have negative results.

Pulmonary tuberculosis of the acute pneumonic type is occasionally

marked by so violent an onset and such rapid consolidation as to make its differentiation from lobar pneumonia impossible until tubercle bacilli or elastic tissue appear in the sputum. In general it may be said that errors in diagnosis are best avoided by frequent and painstaking examinations of the thorax and of the sputum.

Prognosis.—The mortality of lobar pneumonia varies much at different times and in different locations. In general it ranges between 25 and 35 per cent. In children the death rate, in contrast to that of bronchopneumonia, is very low. The mortality is highest among those of advanced years and those debilitated by alcohol or by chronic disease. The extent of lung involvement does not play a prominent part in determining the prognosis. Much more important than the extent of consolidation are the degree of toxemia and the character of the heart action, and these frequently bear no relation to the size of the lesion. The advent of pulmonary edema is always of grave significance. Marked abdominal distention also adds much to the seriousness of the situation.

Such complications as meningitis and malignant endocarditis are always fatal.

Treatment.—No satisfactory specific method of treatment is yet known, and the best results are still obtained from intelligent and watchful treatment of the symptoms as they arise.

The attempts to cut short the disease by venesection or by such drugs as veratrum viride or tartar emetic have been altogether abandoned as useless if not actually harmful.

Absolute rest in bed and freedom from all movement or excitement must be insisted upon. The room should be cool, quiet and thoroughly ventilated and the bed covering and clothing light and comfortable. (See Appendix, p. 582.) The diet should be fluid (milk, broths, gruels, etc.), should be given in small amounts and at frequent intervals and should be carefully watched with a view to the prevention of flatulent distention. A calomel purge at the outset is usually desirable.

In the first days of the disease the distressing PAIN in the side and short, dry COUGH are the symptoms most apt to demand attention. If the pain is not severe it can often be controlled by the use of hot flaxseed poultices or of ice-bags applied to the painful area. If the pain is very severe and is constantly aggravated by the cough, and if the patient is restless and sleepless because of it, then morphine should be given subcutaneously in repeated doses of from $\frac{1}{12}$ to $\frac{1}{6}$ of a grain. The drug at this stage can safely be given and is often of the greatest possible value in relieving the pain and cough, in producing sleep and thus saving the patient's strength. Later in the disease, when dyspnea and cyanosis appear, it should be used only with great caution, because of its effect upon the respiratory center. Fortunately it is then not often needed. For the cough alone codeine (gr. $\frac{1}{8}$–$\frac{1}{4}$) or heroin (gr. $\frac{1}{20}$–$\frac{1}{10}$) may be used. The usual expectorant mixtures have little effect, are apt to disturb digestion, and are therefore to be avoided.

The TEMPERATURE itself does not require treatment unless it be unusu-

ally high or unless cerebral symptoms be marked. It is best reduced by cold packs, alcohol sponge baths or ice-bags to the chest. Tub baths, which have been strongly recommended by some, are open to the serious objection that they cannot be used without causing the patient much disturbance and fatigue. Our best means of combating the TOXEMIA is the introduction of large quantities of fluid which, by diluting the toxins and causing their increased elimination by the kidneys, tend to diminish their noxious effects. This can be done most easily by insisting upon the patient's drinking freely and frequently of water or lemonade. The same purpose is accomplished by the use of subcutaneous infusions of normal (0.8 per cent.) salt solution (Oss–Oj) or of large enemata of the same sort.

THE PROGRESSIVE WEAKENING OF THE HEART is the symptom which demands most attention. It varies greatly in different cases. Some cases require no stimulation whatever and this is especially apt to be true if great care has been exercised in guarding the patient's strength in the early days, by enjoining absolute rest, relieving pain and cough and inducing sleep. In a majority of cases the heart, in the later days of the disease, will require some support and stimulation. For this purpose alcohol is the drug most widely used. It is best administered in the form of whiskey or brandy (℥ss–℥i every 2, 3, or 4 hours) well diluted with water or in milk. While alcohol seems to be of great value in suitable cases, its routine administration in every case cannot be too strongly condemned. Nor is it always necessary or wise to give it in alcoholic subjects. A stimulant of perhaps greater value than alcohol is strychnine. It is given by mouth or subcutaneously in doses of gr. 1/60–1/20 every three or four hours. Much difference of opinion exists concerning the value of digitalis in pneumonia. It has seemed to the writer to be sometimes of great value in critical cases when given in full doses. Strophanthus and caffeine are also used. Concerning the value of oxygen inhalations there is also much conflict of opinion. Where there is much dyspnea and cyanosis they should be tried and persisted in if they do not add to the patient's distress.

Pulmonary edema is always an ominous symptom and should be promptly met by increased stimulation and by the frequent application of dry cups. Meteorism is best relieved by reduction of diet, by frequent hot fomentations to the abdomen and by turpentine enemata. (See Appendix, p. 582.)

SERUM TREATMENT.—Although the critical termination of pneumonia seems due in all probability to the development in the body of an active antitoxin, the use of antitoxic serum obtained from immunized animals has thus far been disappointing and has not yet come into general use.

PROPHYLAXIS.—In view of the infectious nature of the disease it is of great importance that the sputum be disinfected and destroyed with the same care as in tuberculosis. The general observance of this simple precaution would probably do much to reduce the alarming prevalence of this disease.

BRONCHOPNEUMONIA
(Lobular Pneumonia.   Capillary Bronchitis)

Definition.—An inflammation of the finer bronchi and the adjacent air-vesicles, occurring in scattered areas throughout both lungs.

Etiology.—Bacterial infection is regarded as the chief cause of bronchopneumonia, although no specific organism has been found. Pneumococci, streptococci, staphylococci, Friedländer's bacillus, the diphtheria bacillus, Pfeiffer's bacillus of influenza, and other organisms are found in the affected lungs.

The disease occurs in three different forms.

1. Primary bronchopneumonia is met with almost wholly in children under two years of age.

2. Secondary bronchopneumonia is a complication of the acute infectious diseases, especially those in which bronchitis is marked, as measles, whooping-cough, diphtheria, and influenza, less often in typhoid, small-pox and scarlet fever. It also occurs as a terminal complication in bronchiectasis, emphysema, chronic bronchitis, and tuberculosis, and also in chronic nephritis, diabetes and heart disease.

3. Aspiration or deglutition pneumonia is a variety of bronchopneumonia caused by the entrance of foreign material, such as vomitus, pus, blood, necrotic tissue and the like, into the lungs. Thus during anesthesia or in a stuporous or comatose condition vomitus may be aspirated into the lungs. In syphilis, tuberculosis, or cancer of the larynx, pus or necrotic material may be drawn into the lungs, or in abscesses rupturing into the lungs or respiratory tract the bronchi may be filled by pus. Doubtless bacteria rather than the foreign materials are harmful.

Bronchopneumonia is favored by any influence lowering the resistance of the patient. Infancy and old age are therefore the periods of life most affected. In infancy rickets is an important factor, in old age any debilitating disease.

Morbid Anatomy.—Three groups or types represent different stages of the inflammatory process. Both lungs are involved in most cases. (1) The lungs present the lesions of bronchitis and about many of the bronchi there is a narrow zone of deep congestion. Microscopically the bronchi contain mucopus, the walls of the bronchi and some of the adjacent interstitial tissues are infiltrated with leukocytes, and the contiguous air cells contain leukocytes, epithelium, and mucus, rarely fibrin. The posterior surface of the lungs under these conditions shows depressed deep-blue areas of collapse, due to the obstruction of the terminal bronchioles. By inflation with a blow-pipe through the bronchi these depressed areas can regularly be filled out.

(2) The second type shows areas of consolidation about the bronchi throughout both lungs. This peribronchial consolidation appears on section as circular, deep-red areas surrounding the bronchi, rising above the uninflamed parts of the lung, and feeling distinctly firm to the fingers. The bronchi are usually filled by plugs of mucopus. Areas of

collapse are regularly present. Microscopically the process is the same as in the first type except that the consolidation extends more or less widely about the bronchi.

(3) The third type is the pseudo-lobar bronchopneumonia, so-called because the consolidation involves the greater part of both lower lobes. Fibrinous pleurisy often accompanies this extensive consolidation, and the process looks very much like lobar pneumonia. The section, however, presents a mottled or marbled appearance, owing to the fact that the earlier parts of the inflammatory process affecting the bronchi and adjacent alveoli become lighter in color and thus stand out in contrast to the more distant peripheral areas of fresh congestion and consolidation. Often the branching figures of the bronchi can be plainly made out.

The distinctive features of bronchopneumonia are that it centers about the bronchi, the consolidation extending peripherally from them, that the walls of the bronchi and the adjacent interstitial tissue are infiltrated with leukocytes, that the exudate shows more epithelium and relatively fewer leukocytes in the bronchi and air-cells, and that fibrin is less abundant.

Aspiration pneumonia is often marked by suppuration or gangrene in the areas of peribronchial consolidation.

Symptoms.—THE PRIMARY FORM begins with a rise of temperature, possibly a convulsion, followed by cough, increased frequency of respiration, and prostration. The child is drowsy, vomiting may occur, and the bowels are constipated. The physical signs are those of bronchitis. The fever and other symptoms persist about one week. The temperature often falls by crisis, and it is difficult except for the absence of signs of consolidation to distinguish this affection from lobar pneumonia.

THE SECONDARY TYPE develops from acute bronchitis, either itself primary or a part of one of the acute infectious diseases. With the onset of pneumonia, the temperature rises to 104° or 105°, the pulse becomes more rapid, the prostration more marked, the cough more frequent and distressing, but there is no change in the physical signs of bronchitis. Gradually as the process extends all the symptoms become severer, the dyspnea may become extreme, the respirations reaching 80 to 100 in the minute in infants or young children, and inspirations often being marked by sinking of the suprasternal parts, the epigastrium and the lateral parts of the thorax owing to the obstruction of the bronchi by secretion. As the asphyxia increases the child becomes more restless, more stuporous, cyanosis appears and deepens steadily, and profuse cold sweating occurs. After three or four days of the pneumonia, the lungs being generally consolidated, the percussion note over the posterior parts of the chest becomes duller, the respiration is harsh and blowing, and the râles, subcrepitant or loose and sonorous, are abundant. Frank signs of consolidation with dulness, bronchial voice and breathing and increase of vocal fremitus may be had over limited areas, but are rare.

In the elderly, bronchopneumonia very closely resembles that of children, except that the temperature is rarely above 102° or 103°, and the cyanosis and dyspnea are not so severe.

The temperature curve in bronchopneumonia is very variable, usually of distinctly remittent type. In the primary cases the range of fever is moderate, in the secondary type very high temperatures may be recorded. (See Fig. 4.)

PHYSICAL SIGNS.—In the early stages the only pulmonary signs are those of acute bronchitis, subcrepitant or mucous râles; later the percussion note is impaired, especially posteriorly; over both lungs there are areas of harsh, high-pitched breathing, as well as râles, and in some cases definite areas of dulness, bronchial voice and breathing.

COURSE.—In the primary form the duration is short, a week or ten days. The secondary bronchopneumonia is more severe, and, if the patient lives, more protracted, lasting for several weeks, possibly for months. In this form the temperature falls by lysis, the constitutional symptoms disappear and the patient is left greatly enfeebled, and, by reason of the changes in the walls of the bronchi, not infrequently suffers from chronic bronchitis or bronchiectasis.
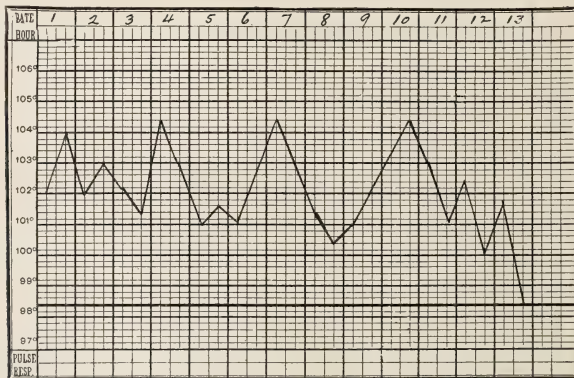
FIG. 4.—Temperature curve in bronchopneumonia.

Diagnosis.—The primary type, especially in children, may be confused with lobar pneumonia. The sudden onset of the latter, the higher and more constant temperature, high leukocytosis, definite signs of consolidation, and termination by crisis should differentiate it. From acute bronchitis bronchopneumonia is often distinguished only by the higher range of fever, greater prostration, longer course, and generally more marked severity. Definite areas of pulmonary consolidation would be decisive.

The secondary type of bronchopneumonia is regularly recognized by the conditions under which it develops, and in which lobar pneumonia is very rarely met.

From tuberculosis the more protracted cases of bronchopneumonia can be differentiated in many cases only by repeated examinations of the sputum. Smears of the pharyngeal mucus may be taken in children who will not expectorate. THE BLOOD shows a moderate leukocytosis, usually

lower than that observed in lobar pneumonia, rarely exceeding 25,000 except in cases of very extensive consolidation.

Prognosis.—Bronchopneumonia is always a grave disease. The mortality is high in infants and the very old. The primary type usually ends favorably. The secondary type is the chief cause of the mortality of many acute infectious diseases, especially measles, whooping-cough, diphtheria and influenza. Few fatal cases of these diseases occur in which bronchopneumonia does not play a large part.

Treatment.—Prophylaxis. Proper treatment of the acute infectious diseases of which bronchopneumonia may be a complication will prevent many cases. The general condition of children in hospitals and asylums has much to do with the frequency and mortality of this type of pneumonia. Improvement, brought about by good food, fresh air, and attention to hygiene will diminish both. Cleansing of the mouth by mild antiseptic washes is important in all diseases likely to be complicated by pneumonia.

The treatment of cases of primary bronchopneumonia must be carried out on the same principles as that of lobar pneumonia. Rest in bed, a fluid or soft diet, evacuation of the bowels by enema, if necessary, may be all that is required for mild cases. All patients should be carefully fed with milk, broths, gruels, and soft solids. Water should be given freely and fresh air supplied, no matter what the temperature.

In the severer cases there are two chief indications: to control the bronchitis and to support the heart. Careful feeding and fresh air are essentials. Medicines have little or no influence on the bronchitis. Light poultices of flaxseed meal applied over the whole anterior or posterior chest and renewed every two or three hours often give relief to the cough, dyspnea and cyanosis. Priessnitz applications, made by dipping a layer of flannel in water at 70°–75° F., then wringing it out, applying it to the chest and covering it with several layers of dry flannel, may be used instead of the poultice. Such applications may be repeated every three or four hours. In some cases a jacket of cotton covering the entire chest and kept continuously applied is satisfactory. In adults dry cups may be used for the sake of counterirritation. Expectorant mixtures of ammonium carbonate or chloride and syrup of ipecacuanha are generally recommended, but they are nauseating and of doubtful value. For high fever and restlessness a tepid sponge bath, the water at a temperature of 90° to 95°, is the best remedy. Antipyretics of the coal tar class should not be used. For the support of the heart stimulants may be called for. Whiskey is probably the best of these and may be given freely in milk or other liquids. Digitalis may be given by mouth, and strychnine either by mouth or hypodermatically.

When cyanosis results from the accumulation of secretions in the bronchi, relief may be had by an emetic dose of ipecac or by alternate douching of the chest with hot and cold water to excite coughing and deepen respirations. Opium should be given only in emergency to relieve pain or distressing cough, as it tends to promote the accumulation of

secretions which should be gotten rid of. Although children do not expectorate, the cough expels the secretions from the bronchi into the pharynx and they are then swallowed.

Convalescence from bronchopneumonia is likely to be slow and difficult. At this time a change to a milder climate is often the best of treatment.

CHRONIC INTERSTITIAL PNEUMONIA

Sclerosis or Cirrhosis of the Lung.   Chronic Fibrous Pneumonia

Definition.—A chronic inflammation of the pulmonary interstitial tissue, resulting in increase of fibrous tissue, either local or general.

Etiology.—Any lesion of the lung, traumatic or inflammatory, may be followed by a sclerosis, which may be classified under this heading. The sclerosis is usually localized, but may extend throughout the affected lobe. The cases of clinical importance regularly follow (1) lobar pneumonia; (2) bronchopneumonia in rare cases causing disseminated sclerosis of the lung; (3) pleurisy; (4) pressure of aneurisms or other tumors upon the lung; (5) syphilis; (6) inhalation of irritating dust of coal, iron, or stone. These cases are described later under Pneumoconiosis.

Morbid Anatomy.—The essential feature is the increased fibrous tissue. (1) In the disseminated form the fibrous tissue is thickened and increased about the bronchi, or arteries, or in strands running from the pleura or interlobular septa. The bronchi are usually dilated and the intervening lung emphysematous. (2) The massive form affects one lobe or one lung. The pleura is greatly thickened and adherent. The lung or lobe is contracted into a shrivelled mass of dense tissue which is firm, grayish-white, cuts with resistance and on section shows a dense mass of fibrous tissue surrounding bronchi which are chronically inflamed and usually dilated, so as to form cavities of some size. The heart is regularly displaced toward the affected side in these cases, and the right ventricle dilated.

Symptoms.—These are produced not by the sclerosis of the lung itself, but by the chronic bronchitis and bronchiectasis resulting from it. There are chronic cough, expectoration, and possibly dyspnea. These symptoms vary with the course of the bronchitis, improving in summer, becoming worse in winter. If the bronchiectasis is marked the patients have the paroxysmal cough and expectoration characteristic of that affection. Hemorrhage from the lungs is not uncommon. Finally the right ventricle becomes dilated and the clinical picture is complicated by the signs of cardiac failure, increasing dyspnea, cyanosis, and edema.

PHYSICAL SIGNS.—In the disseminated type the signs are those of bronchitis or bronchiectasis. In the massive or lobar type very characteristic signs are produced by the retraction of the lung. The affected side is shrunken and smaller, the intercostal spaces narrowed or obliterated by overlapping of the ribs. The spine may be curved. The heart

is drawn toward the affected side, and the cardiac impulse may be extensive and forcible owing to exposure of the heart by the retraction of the lung. Expansion may be practically absent. The affected area is dull or even flat. On auscultation the râles of the bronchitis or bronchiectasis and varied changes in the respiratory murmur may be heard, diminished or intensified breathing, amphoric or even cavernous sounds. The vocal fremitus is increased or diminished. The heart may give signs of dilatation and hypertrophy with murmurs.

Course and Prognosis.—The affection is chronic and incurable, but except in extreme cases consistent with many years of life.

Diagnosis.—The disease can be distinguished from tuberculosis only by the absence of tubercle bacilli from the sputum.

Treatment.—The only treatment is that adapted to the condition of the bronchi and the heart. (See pp. 7 and 183.)

PNEUMOCONIOSIS

Definition.—A special form of interstitial pneumonia produced by the long-continued inhalation of irritating dust, especially that from coal, iron, stone, and the like. The disease has received various names, depending on the form of dust concerned, such an anthracosis (coal-dust), siderosis (iron-dust), chalicosis (stone-dust), kaolinosis (clay-dust), but varies little except in the color of the affected lungs.

Etiology.—The affection is an occupation disease, brought about by working for years in the dust-laden atmosphere of mines, or work-shops of special character. Coal-mining and the grinding of sharp-edged tools, the making of millstones, grain-shovelling and the like are all hazardous occupations. Ordinarily dust inhaled is stopped in the nasal chambers, or if it reaches the bronchi is checked there by the action of the mucus and the ciliated epithelium. Only when these defensive provisions are overwhelmed does it pass through the lining of the finer bronchi and the alveoli to reach the pulmonary tissue. Thence the dust is carried by leukocytes and the lymph-stream (1) to the lymph-nodules about the arteries and bronchi; (2) to the interlobular septa; (3) to the bronchial, tracheal, and substernal lymph-glands. Wherever the dust is deposited in quantity the tissues are correspondingly pigmented.

Morbid Anatomy.—The lungs regularly are more or less generally pigmented; in the case of miners they may be almost coal-black. The tracheal, bronchial, and mediastinal glands are likewise pigmented. The lungs also present the lesions of chronic bronchitis and emphysema and disseminated patches of fibrous tissue, due to chronic interstitial inflammation. In some cases, the fibroid areas break down and leave cavities of various size, full of mucopurulent material containing the dust.

Symptoms.—After some years of working in the dusty atmosphere the patients develop a chronic cough and expectoration (chronic bronchitis). The sputum is pigmented, black or gray, and mucopurulent in

3

character.   Later with the development of emphysema the workers suffer from dyspnea and possibly cyanosis.

PHYSICAL SIGNS.—The patients present the signs of chronic bronchitis and emphysema, in rare instances, of consolidation with cavities.   The X-rays show a diffuse even sclerosis of the lung, suggestive but not diagnostic.   Small foci not distinguishable from those of tuberculosis regularly appear in the plates.   (See Fig. 5.)

FIG. 5.—Siderosis of the lung in a granite cutter. Compare the diffuse mottling (or mackerel effect) of both lungs with the normal left lung shown in Fig. 6.

Course and Prognosis.—The disease is chronic, lasting for years. The duration of life is shortened, in some cases very notably.   Few grinders of cutlery live beyond forty, and life in the mines is relatively short.

Diagnosis.—The conditions under which the affection develops make the diagnosis easy.   Tuberculosis must be excluded by the absence of the constitutional symptoms and failure to find bacilli.

Treatment.—Adequate ventilation in mines and working rooms and the use of respirators diminishes the frequency of the disease.   Change of occupation is, of course, desirable for those affected.   Otherwise the treatment is that of the chronic bronchitis and emphysema.

EMPHYSEMA

Definition.—Pulmonary emphysema is a dilatation of the infundibula and alveoli of the lungs, with thinning or atrophy of their walls. Several forms are described:

(1) COMPENSATORY EMPHYSEMA.—Whenever any part of the lungs is prevented from expanding in inspiration, there occurs a compensatory distention of the remaining parts. In some cases the emphysema so caused is local, about tuberculous foci for example, usually it is general. Thus when one lung is bound down by adhesions or compressed by pleural effusions or tumors, there develops a marked distention of the other side. With persistence of the condition the walls of the alveoli atrophy and the emphysema becomes permanent. Compensatory emphysema occurs particularly in tuberculosis, pneumonias, especially unilateral chronic interstitial, hydrothorax, empyema and the like.

The affected side of the thorax looks large, and appears to expand more fully than normal, both these features being accentuated by the limitation of motion and possibly retraction of the lung primarily diseased. The percussion note is exaggerated, the voice and breathing intensified, the vocal fremitus increased. With removal of the cause, the compensatory emphysema usually disappears.

(2) HYPERTROPHIC EMPHYSEMA.—In this form we have to do with a general dilatation of the infundibula and alveoli of both lungs, especially their anterior parts. By reason of the increased size of the lungs it has also been called the large-lunged emphysema.

Etiology.—Two factors are said to be operative:

1. A congenital defect in the elastic tissue of the lung, a condition not proven but assumed to exist to explain the fact that the condition develops in only a few of those subjected to conditions calculated to cause it. Transmission of the disease in families favors this view.

2. Increased intrapulmonary pressure, especially that caused by holding the breath and straining during expiration, such as occurs in pertussis, asthma, and other severe coughing, or in blowing musical instruments, blowing glass, and the like.

The disease may develop in childhood, but is commonly seen in adult life, and in men rather than women.

Morbid Anatomy.—The lungs are large and full, frequently covering the pericardium and meeting in the mid-line, when the thorax is opened. They do not collapse, are light in weight, pale in color, pit upon pressure, and in most cases the over-distended air-vesicles can be clearly seen on the surface. On section the lesions of chronic bronchitis, and possibly of bronchiectasis, are found. Microscopically there is a thinning of the alveolar walls and in places many of the air-vesicles fuse into one large bulla. When these changes are marked they lead to a considerable reduction in the capillary exposure in the walls of the alveoli and in part account for the marked cyanosis. The elastic tissue in the alveolar walls shows signs of degeneration and in places may have altogether disap-

peared. The bronchi show chronic inflammation and possibly dilatation. The heart shows hypertrophy and dilatation, especially on the right side, and there may be chronic congestion of the liver, spleen, kidney, and other viscera as the result of obstruction to the pulmonary circulation.

Symptoms.—The marked symptoms of emphysema are cough, dyspnea, and cyanosis. (1) The cough is due to the associated chronic bronchitis or bronchiectasis, and as in those conditions it is regularly better in summer than winter, and all the symptoms are greatly aggravated by fresh attacks of bronchitis. The expectoration is mucopurulent in character, and may be either scanty or abundant. (2) The dyspnea is due largely to the failing elasticity of the lung and is therefore mainly expiratory, resembling that of asthma, with which emphysema is frequently associated. The dyspnea at first is slight, and occurs only on exertion, increases gradually, becomes persistent and severe in the advanced stages. Inspiration is then short and jerky, expiration prolonged and difficult and accompanied with much wheezing. (3) Cyanosis in the early stages is slight and transitory, later it becomes constant and very marked. Emphysema is one of the few conditions in which, with deep cyanosis of the lips, face and extremities, the patient can still go about with comparative comfort. Congenital heart disease or poisoning with one of the coal-tar preparations may present a like picture. With advanced emphysema the patient usually appears thin, though muscular. In the final stages the right heart fails and symptoms of broken compensation are present. Hemoptysis is not infrequent in the course of the disease.

PHYSICAL SIGNS.—The chest is deepened antero-posteriorly, giving it the barrel shape; the shoulders are round; the curve of the spine increased, the clavicles prominent, the anterior chest bulging, the supraclavicular fossæ marked. The costal cartilages are rigid and in inspiration the thorax rises rigidly like a cuirass. On percussion the note is exaggerated, or drum-like. The area of pulmonary resonance is increased, so that the cardiac dulness is diminished or obliterated, and the lower limits of lung resonance are found at the sixth or seventh rib in front and the eleventh or twelfth behind. Liver dulness is correspondingly lowered and splenic dulness usually absent. On auscultation the inspiratory murmur is short and weak, expiration is prolonged and high-pitched. Usually there are abundant sibilant, sonorous, or mucous râles from the bronchitis, especially posteriorly. Dilatation of the right heart may be shown by marked epigastric pulsation, pulsation of the veins of the neck, and dilatation of the superficial veins.

Diagnosis.—The physical signs are characteristic of emphysema, and cannot well be mistaken.

Course and Prognosis.—The disease is progressive, but slow; and under favorable conditions life may be prolonged for many years. The patients succumb to failure of the right heart or to bronchitis and bronchopneumonia.

Treatment.—For the emphysema, little can be done directly. The chief indications lie in the treatment of the chronic bronchitis and avoid-

ance of fresh attacks. For these ends a mild, dry climate, such as that of Southern California, the Riviera, or Egypt, is desirable during the winter months. Attention must be given to the diet and clothing and the general health of the patient. With exacerbations of the bronchitis the patient must be put to bed and treated as for acute bronchitis. For extreme cyanosis and dyspnea arising under these conditions, venesection is indicated. Withdrawal of a pint of blood from the arm may save the patient when all other measures fail. The efficacy of this measure lies in the relief afforded the overburdened right heart.

ATROPHIC EMPHYSEMA

Senile or small-lunged emphysema is properly an atrophy of the lung, occurring as part of the general atrophy of the tissues in old age. The chest is small, and the ribs more oblique than normal. The lungs are small and collapse on exposure. The intervesicular septa are atrophied and many alveoli fused into large bullæ. Atrophy of the capillaries in the septa naturally accompanies the process. The patients suffer from winter cough with more or less dyspnea for many years.

ACUTE VESICULAR EMPHYSEMA

Under certain conditions of dyspnea marked by strong inspiratory efforts, and possibly obstruction of the bronchi, the lungs may be acutely distended and remain so for some time. This condition gives some of the physical signs of emphysema, enlarged areas of pulmonary resonance, an exaggerated percussion note, and many sibilant or sonorous râles with prolonged expiration, but there is no atrophy of the elastic tissue and no permanent emphysema. The condition may develop rapidly in certain cases of bronchopneumonia, cardiac dyspnea, or angina pectoris.

INTERSTITIAL EMPHYSEMA

This form of emphysema is produced by rupture of air-vesicles of the lungs during strong expiratory efforts, such as occur in whooping-cough, bronchopneumonia, parturition, or heavy lifting. Postmortem the air is found in large blebs in the interstitial tissue and beneath the pleura, especially at the roots of the lungs or along their anterior margins. Rupture through the pleura may cause pneumothorax, or in some cases the air makes its way through the mediastinum into the neck or even the arms, appearing as subcutaneous emphysema of these parts.

ABSCESS OF THE LUNG

Etiology.—Abscess of the lung is never primary but may result from several conditions.

1. Infected wounds such as those produced by broken ribs or by bullets.

2. Abscesses in neighboring organs, the liver, the mediastinum, the

pleura, or the peritoneum, may by extension produce suppuration in the lung, or may rupture through it.

3. Inflammatory processes in the lung may terminate in suppuration. (a) Lobar pneumonia frequently results in an abscess occupying more or less of a lobe. (b) Bronchopneumonia, especially the aspiration pneumonia produced by the inspiration of septic material of any kind, gives rise to one or more abscesses of smaller size. (c) Tuberculosis in its advanced stages may produce large abscesses of the lung.

4. Multiple metastatic abscesses of the lungs are frequently met with in pyemia of all forms, in malignant endocarditis, and the like.

5. New growths of the lung are often complicated by suppuration and abscess formation either in the area of the growth or adjacent parts.

Morbid Anatomy.—The multiple abscesses of pyemia, or aspiration pneumonia, are numerous and of small size. The abscesses following lobar pneumonia, or infected wounds, or produced by extension, are usually single and may involve the greater part of a lobe or a lung. Tuberculous abscesses or cavities may be of any size or number. The contents of the abscess are pus, usually greenish-gray in color, and foul-smelling, but not so offensive as in gangrene or putrid bronchitis. Elastic tissue from the broken-down lung may be found in it. Bacteria of various kinds may be present. In the pus discharged from abscess of the liver the ameba has been found.

Abscesses of the lung are regularly complicated by pleurisy, which may be purulent, or the abscess may rupture through the pleura and thus give rise to empyema. Most often the abscesses rupture into a bronchus and the pus is discharged in this way.

Symptoms.—The multiple abscesses of pyemia give no distinctive symptoms and are usually not recognized during life. In other cases the patients present fever, continuous or of remittent type, sweating, rapid pulse, cough, dyspnea, and pain in the chest. These symptoms are engrafted on those of the original condition, such as bronchopneumonia or lobar pneumonia. Sooner or later the pus is expectorated. Often the abscess ruptures suddenly and the patient coughs up a large quantity of foul pus at once. A leukocytosis is present except in tuberculous cases. The secondary anemia is usually marked.

The PHYSICAL SIGNS are those of a localized collection of fluid or, after the rupture of the abscess, of cavities. Thus after lobar pneumonia dulness persists, usually with a gradual loss of the intense bronchial voice and breathing and vocal fremitus. If the abscess is large and close to the pleura the signs may be those of empyema, and only the depth at which the pus is found on exploration or operation locates the process as pulmonary. In the bronchopneumonic cases the abscesses are smaller and much more difficult to locate.

Course and Prognosis.—The course is usually very protracted. Many of the cases are fatal. If the abscess discharges through a bronchus or is drained, recovery may take place by gradual obliteration of the cavity, usually requiring many months.

Diagnosis.—This is often not made except at autopsy. In some cases the combination of the constitutional symptoms of suppuration and the expectoration of large amounts of pus make the diagnosis easy. The demonstration of elastic tissue in the pus is proof of destruction of the lung. Practically the cases of greatest difficulty are those following lobar pneumonia, where the persistent fever and physical signs suggest delayed resolution, abscess of the lung, or empyema, or tuberculosis. In these cases repeated exploration of the chest with a large calibered
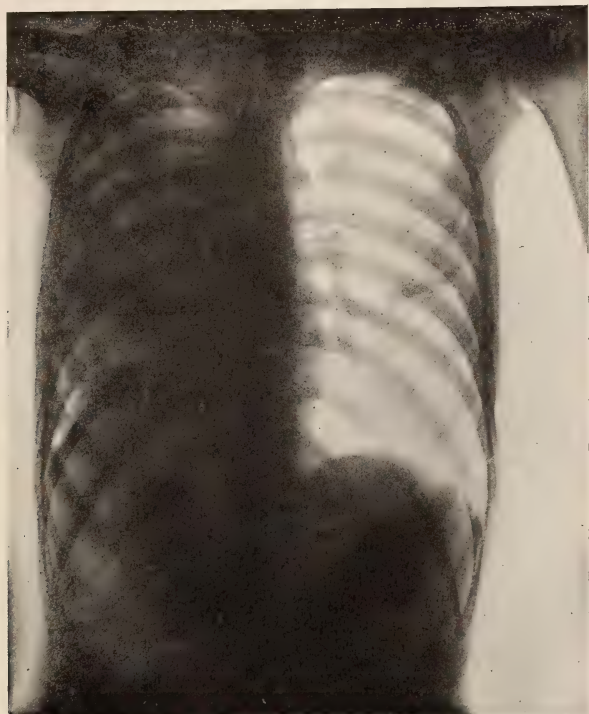
Fig. 6.—An abscess of the lung occupying most of the left chest, obscuring the cardiac outlines and darkening the whole area of the left lung.

needle is essential. At times only the depth at which the pus is found indicates its location in the lung. Occasionally the sudden discharge of pus through a bronchus clears up the diagnosis. The blood in these cases regularly shows a leukocytosis usually accompanied by an increase in the percentage of polynuclears.

The X-rays show a heavy shadow on the affected side, rarely sharply defined, and not distinguishable by these means alone from pleural effusions, thickening of the pleura, or consolidation of the lung. (See Fig. 6.)

Treatment.—Careful feeding and nursing help to maintain the patient's strength. No medicinal treatment is known to affect the process

in the lung. When an abscess is discharging postural treatment may be of value, the patient lying flat upon the bed with the foot raised, or upon one side or the other, as is found to best drain the abscess, for some hours each morning, until the cavity is emptied of its contents. He may then be allowed to assume an ordinary position. When an abscess is located by the exploring needle, drainage by surgical intervention is required. In some cases where the abscess is being imperfectly emptied through a bronchus, operation may be necessary.

GANGRENE OF THE LUNG

Etiology.—Gangrene of the lung is never primary, but complicates some previous affection of the lung, bronchitis, bronchopneumonia, lobar pneumonia, or abscess, or embolism of the pulmonary artery. Why gangrene develops in some cases and not in others is not known. Lowered tissue resistance is the most plausible suggestion. Gangrene of the lung is relatively rare, but is most often seen following (1) aspiration bronchopneumonia or the bronchopneumonia complicating noma; (2) bronchiectatic or tuberculous cavities; (3) lobar pneumonia; (4) embolism of the pulmonary artery, when the embolus has come from a gangrenous focus; (5) in debilitating diseases, such as typhoid fever, diabetes, and the like.

Morbid Anatomy.—The gangrenous area of the lung may be large or small, single or multiple. As usually seen the affected portion presents a central cavity containing a dark, putrid fluid, its walls composed of soft, ragged, greenish-black material, beyond which is a more or less extensive area of pneumonic consolidation. Pleurisy or empyema is associated, sometimes pyopneumothorax. Abscess of the brain is not infrequently found as a complication.

Symptoms.—The inception of gangrene is preceded by the symptoms of the primary affection of the lung. The development of gangrene is marked by an increase in severity of the general symptoms and the appearance of the characteristic sputum. The expectoration and breath are intensely fetid, such as are met with in no other condition except putrid bronchitis. The expectoration is abundant, as a rule, and the patient is harassed by a constant cough, as well as the frightful taste and odor of the secretion. In children with noma, the expectoration being absent, and the breath already contaminated by the condition of the mouth, there is no conclusive evidence of gangrene of the lung, yet it is often present. The constitutional symptoms usually become those of intense sepsis and the patients promptly succumb. In some cases, however, the course is less acute and recovery is possible.

Physical Signs.—These are those of the underlying pulmonary condition, bronchitis, pneumonia, or infarction. In advanced cases cavities may be made out.

Diagnosis.—This must be based on the odor of the breath and sputum, and the character of the latter. If collected in a glass, the sputum sep-

arates into three layers, the lowest of greenish-black granular material, the middle a thin dirty fluid, and the top of thick, dark, frothy mucus. Microscopically it shows granular material, fatty crystals, quantities of bacteria, and usually elastic tissue.

Treatment.—This, as in abscess of the lung, must be mainly supportive. Sprays or inhalations of carbolic acid, creosote, and the like, may be used to mitigate the odor. A few cases of successful drainage of gangrenous areas in the lung have been recorded.

NEW GROWTHS OF THE LUNG

Benign tumors of the lung are exceedingly rare. Adenoma, fibroma, chondroma, and osteoma are possibilities. Malignant tumors occur as carcinoma or sarcoma. Primary new growths of this class are exceedingly rare.
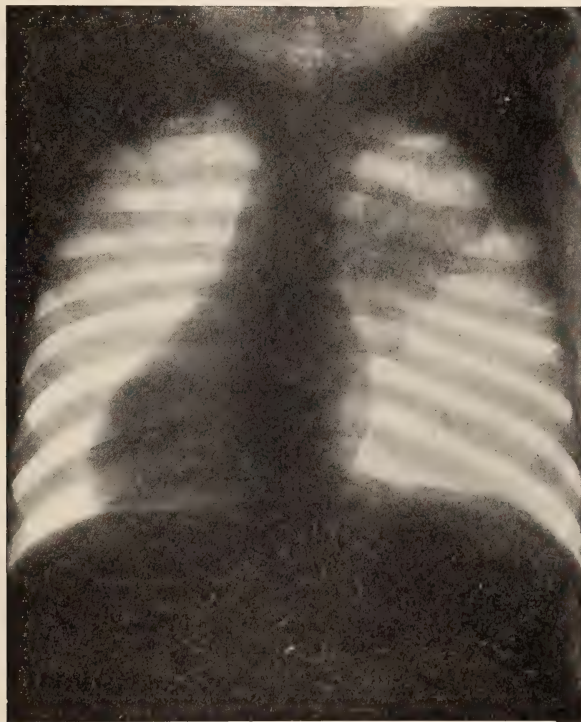
Fig. 7.—An intrathoracic growth giving an extensive shadow above and to the right of the heart. The growth gave no physical signs, but produced attacks of dyspnea closely resembling bronchial asthma. It was probably a sarcoma of the mediastinal nodes and lung.

Carcinoma occurs secondarily to primary growths in the breast, glands, esophagus, or stomach, or other viscera. Sarcoma most often follows sarcoma of bone, but the primary growth may be in any part. Carcinoma is rarely met with under 40. Sarcoma may occur at an earlier age.

Morbid Anatomy.—The new growths are single or multiple masses

of varying size, whitish or gray in color, compressing the lung or adjacent structures in their growth, frequently involving the pleura and the chest wall.

Symptoms.—An interval of months or years following the primary growth may elapse before the lung is involved. The symptoms are slowly developed. These are thoracic pain, cough, increasing dyspnea, possibly hemoptysis, and eventually cachexia. The symptoms grow progressively worse. Life is rarely prolonged over 6 or 8 months after evidence of pulmonary involvement.

The physical signs are uncertain. A localized area of dulness with diminished voice and breath sounds may be made out. Pleurisy with hemorrhagic effusion is common. In the pleural fluid cells with mitotic figures may be found. A radiogram may be very helpful in deciding the diagnosis. (See Fig. 7.)

Treatment is purely palliative. Morphine may be given to relieve pain and distress.

DISEASES OF THE PLEURÆ
ACUTE PLEURISY

For purposes of description acute pleurisy is classified according to the character of the exudate as fibrinous or dry, serous or purulent. There are no strict lines of division between these forms. Fibrinous pleurisy may become serous or even purulent. For the most part the etiology of these forms is the same. Whether in any given case of pleurisy the exudate shall be fibrinous, serous or purulent, seems to depend in large measure upon the general condition of the patient at the time. We separate them only for convenience of description.

Etiology.—(1) Primary: Pleurisy arises at times without apparent cause. A certain number of these cases are regarded as rheumatic manifestations. Exposure to cold or wet is usually given as the cause, but the more these cases are followed, the greater the number found to be due to bacterial infection, especially to the tubercle bacillus. (2) Secondary to disease of the lung, pneumonia, tuberculosis, cancer, abscess, gangrene, or infarction. (3) Secondary to disease of adjacent organs, the pericardium, peritoneum, mediastinal glands, or esophagus, the vertebræ, ribs, or sternum. (4) Secondary to acute infectious diseases, especially rheumatic fever, or to chronic nephritis, gout, or diabetes.

Bacteriology.—The possibility of pleurisy being due to other causes than bacterial infection must be admitted, but in the great majority of cases where investigation of the question is possible bacteria are found. The tubercle bacillus, pneumococcus, and streptococcus are by all means the most common of these, but a number of others, the staphylococcus pyogenes, streptococcus capsulatus, Friedländer's bacillus, influenza bacillus, the typhoid bacillus, and many others have at times been found, either alone or in association. Any of these organisms may cause a dry, serous, or purulent effusion.

Fibrinous pleurisy, however, most often yields the pneumococcus or streptococcus, the serous effusion the tubercle bacillus, and the purulent effusions the pneumococcus or streptococcus. The demonstration of the tubercle bacillus often requires the inoculation of the suspected fluid into a susceptible animal such as the guinea-pig.

FIBRINOUS OR DRY PLEURISY

Morbid Anatomy.—The pleura is lustreless, opaque, granular on the surface, and slightly swollen or covered with a more or less thick layer of fibrin. Both layers are involved. Microscopically the pleura shows the usual changes of acute inflammation. Following such a pleurisy the membrane is left thickened and more or less adherent.

Symptoms.—Pain in the side, especially on deep respiration, or motion, cough with a slight mucous expectoration or entirely dry, and some increase in respiration, possibly dyspnea, mark the onset of dry pleurisy. Fever is often absent, but the temperature may rise to 100°–101°. The pulse is not rapid and the constitutional disturbance is slight.

PHYSICAL SIGNS.—The characteristic sign is a dry friction rub, caused by the play of the roughened layers of pleura one upon the other, heard during both inspiration and expiration, and seemingly close to the surface. In some cases crepitant or subcrepitant râles only are heard, and these are with difficulty distinguished from like râles arising within the lungs.

Course and Prognosis.—The primary cases last but a few days and the patients recover within a week. That at least one-third of these cases give tuberculin reactions and that 5 or 10 per cent. of them will later develop tuberculosis must be remembered. The course of the secondary cases will depend upon the underlying cause. The blood shows a moderate leukocytosis (10,000–15,000) in most cases.

Treatment.—Limitation of the motion of the affected side by a tight bandage or by strapping with adhesive plaster usually relieves the pain. Instead an ice-bag or a hot poultice may be used. In some cases the severity of the pain requires the administration of codeia or morphine. In convalescence the possibility of later tuberculosis must be remembered.

SEROUS PLEURISY
(Pleurisy with Effusion)

Etiology.—Pleurisy with serous effusion may arise from any of the causes already cited. It is especially common as an apparently primary process, but more than 50 per cent. of these cases can be proven to be tuberculous, and at least 30 per cent. of the patients go on to develop other evidences of tuberculosis.

Morbid Anatomy.—The pleura itself has the appearances described for the fibrinous form. In addition there is present a more or less abundant exudate of serum. This is regularly a clear yellowish fluid with floc-

culi of fibrin, alkaline to litmus, having a specific gravity of 1015 or over, and containing considerable quantities of albumin. Microscopically the fluid shows fibrin, leukocytes, both polynuclear and mononuclear, and endothelial cells. The lung is more or less compressed by the effusion and may be reduced to an airless mass occupying only the upper and posterior part of the thorax. With large effusions, the heart may be displaced to the opposite side, the diaphragm and liver or spleen depressed.

Symptoms.—The onset is usually insidious, but may be abrupt. The disease may begin with a sudden fever, possibly a chill, cough, dyspnea, and pain in the side suggesting a pneumonia. On the other hand, the onset is often so gradual that large quantities of fluid have collected before the patient comes under observation. The chief symptoms are then (1) fever of moderate degree, rarely exceeding 103°, remittent in type and lasting from one to three weeks (see Fig. 8); (2) marked pros-
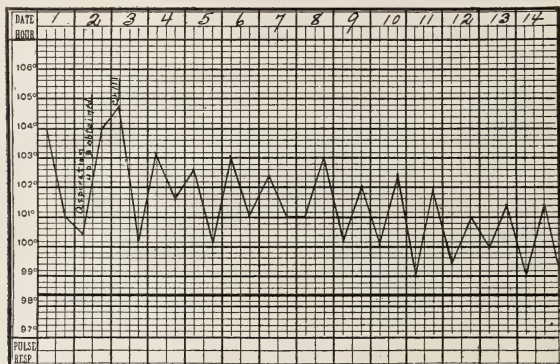
Fig. 8.—Temperature of pleurisy with effusion.

tration with moderate increase in the pulse; (3) a cough with mucous expectoration; (4) dyspnea varying with the quantity of the effusion, very slight and appearing only on exertion in mild cases, so severe as to require the upright position (orthopnea) and associated with cyanosis when the effusion is large; (5) pain in the side is usually present at the onset, disappears with the accumulation of the fluid, and returns upon its subsidence.

PHYSICAL SIGNS.—These must vary with the amount of the effusion. With small effusions the signs are limited to a slight diminution in the respiratory expansion of the affected side, with dulness over the fluid, usually at one base behind, and diminution in the voice, breathing, and vocal fremitus over the same area. Otherwise the examination may be negative. With large effusions very striking physical signs may be developed.

INSPECTION.—The affected side of the chest is nearly immobile, the only motion appearing at the apex in front. It looks larger than the

other and a difference of 1–2.5 cm. may be found on measurement. The lower intercostal spaces behind and in the axilla may bulge. The apex beat may be displaced toward the opposite side.

PALPATION confirms the immobility of the chest, the prominence of intercostal spaces, and the displacement of the apex beat. The vocal fremitus is diminished or lost below the level of the fluid. The transition is not sharply marked.

PERCUSSION.—Below the level of the fluid there is dulness, increasing to flatness at the base in the case of large effusions. Above the fluid in the back the note is still dull from compression of the lung. In front it is normal, or dull, or tympanitic. A dull tympanitic note obtained on percussion beneath the clavicle or lower anteriorly is spoken of as Skoda's
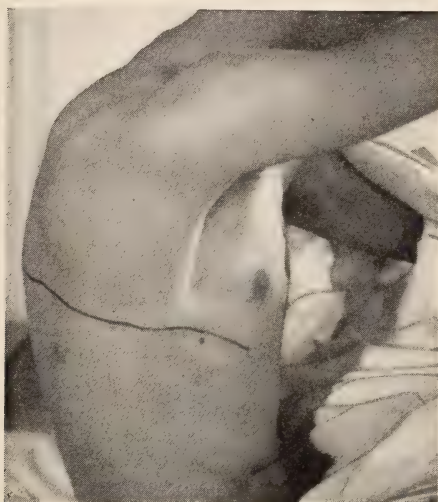
FIG. 9.—The line of dulness in pleurisy, with effusion sloping downward and forward parallel to the ribs.

resonance. The tympanitic note normally obtained on percussion of the area between liver and spleen (Traube's semilunar space) may be obliterated. The line of dulness marking the upper level of the fluid is not horizontal but curved with its highest point usually in the axilla, falling gradually toward both spine and sternum, the forward end usually being the lower. This line is commonly described as the S-shaped line of dulness of Ellis, but it has a single not a double curve. (See Fig. 9.)

Percussion on the sound side frequently discloses a triangular area of dulness adjacent to the vertebral column, the apex of which is at a point on the spine corresponding to the level of the fluid in the other chest, the base of it on the line of the lower level of the pleura, the length of the base line varying from 1 to 3 inches. This area of dulness on the sound side (Grocco's paravertebral triangle, see Fig. 10) is attributed to the distention in that direction of the pleural sac containing fluid,

and it is claimed that the height of the triangle is an indication of the level of the fluid. The sign is not always to be elicited.

AUSCULTATION.—Below the level of fluid the respiratory and voice sounds are usually diminished or lost, above the level they are preserved, and may be normal, diminished, or accentuated. Some notable variations are met with. At times the voice and breathing below the level of the fluid may be definitely bronchial in character, very suggestive of the signs of pneumonia, although nearly always more distant and feeble than in
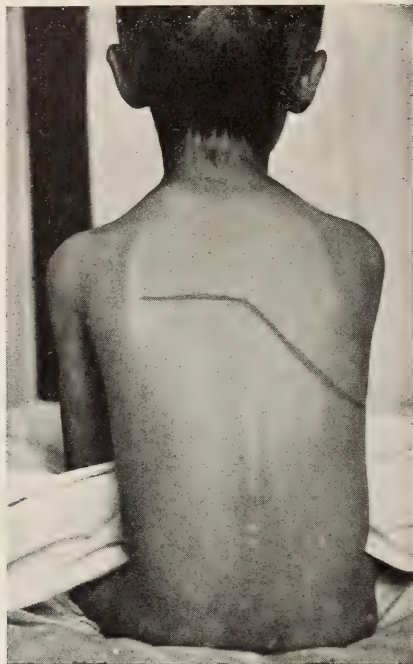
Fig. 10.—Pleurisy with effusion on the left side. Grocco's triangle on the right.

pneumonia. Near the level of the fluid the voice often has a curiously nasal twang, to which the name of egophony has been very inappropriately given.

Above the fluid pleural friction rubs or râles may be obtained, and there is no doubt that similar signs are sometimes obtained through fluid, although this should theoretically be impossible.

The sound lung often develops a compensatory emphysema, appears distended, and gives an exaggerated pulmonary resonance on percussion with corresponding changes of voice and breathing. With the absorption or removal of the fluid the dulness on the affected side diminishes, but does not entirely disappear for weeks or months. At this time the coarse friction rub or râles of dry pleurisy may be heard. Limited expansion on the affected side and diminution of voice and breathing sounds, as well as the dulness, may persist long after the patient is apparently well.

Course.—Many of the cases terminate promptly within two weeks; others are protracted for many weeks or months. The tuberculous cases are usually longer, and in some instances the patients go on to develop the signs and symptoms of pulmonary tuberculosis.

Prognosis.—Death in pleurisy with effusion is unusual, but does occur. It is particularly likely to follow some sudden change of position or the removal of the fluid.

Diagnosis.—In the mild cases unattended by pulmonary symptoms pleurisy with effusion may be easily overlooked. With definite physical signs hydrothorax, empyema, lobar pneumonia, or the thickened pleura and adherent lung of previous pulmonary affection must be distinguished.

Hydrothorax is usually recognized by the conditions under which it occurs, as part of a general anasarca due to heart, kidney, or other disease, and by the absence of fever. The fluid in hydrothorax regularly shows a specific gravity below 1015, a less amount of albumin, and a cellular content chiefly of endothelial cells.

Pneumonia is distinguished by the sudden onset with the initial chill, higher fever, greater prostration, the flushing of the face, the greater restlessness and distress, the more profuse and sometimes blood-tinged expectoration. The bronchial voice and breathing of lobar pneumonia are usually louder and closer to the ear than in pleurisy with effusion and the vocal fremitus in the latter is diminished or lost. The leukocyte count in pleurisy with effusion is rarely above 15,000, in pneumonia it is commonly above. Finally in some cases the exploring needle is required to determine the presence or absence of fluid.

Empyema is most often a sequel of pneumonia. The fever is higher, sweating more marked, prostration and emaciation more profound. The physical signs are practically the same, except that displacement of the heart is more likely to be marked in empyema. Empyema will regularly show a leukocytosis above 15,000, often with an increased polymorphonuclear percentage. The exploring needle is regularly required to complete the differentiation.

The absence of constitutional symptoms usually enables us to recognize the conditions resulting from previous pulmonary disease, such as a thickened pleura with adhesions, but in other cases exploration may be required. Radiographic examination of the thorax may be of value.

The BLOOD in pleurisy with effusion shows in most cases a leukocytosis between 10,000 and 15,000, very rarely above the latter figure. Often there is no leukocytosis, and in the tuberculous cases there may be a leukopenia. The latter cases may also show a differential lymphocytosis. A moderate secondary anemia is regularly present.

Pleural Fluid.—The exudate in pleurisy with effusion is ordinarily a clear yellowish serum, alkaline to litmus, of a specific gravity of 1015 or higher, and containing much albumin. The fluid may contain flakes of fibrin, may coagulate spontaneously, and is frequently tinged with blood drawn either from the pleura or lung by the needle. Effusions which are permanently bloody are always highly suggestive of tuberculous

or cancerous processes.   The *cytology,* that is, the differential counting of the cells to be found in the exudate, is sometimes of help in diagnosis. Widal's formulæ are as follows:—(1) a predominance of polynuclear leukocytes means an effusion of infectious origin (pneumococcus, strepto-coccus, staphylococcus); (2) of lymphocytes, a tuberculous effusion; (3) of endothelial cells, especially in plaques or sheets, of mechanical origin (hydrothorax).   In cancer, cells with mitotic figures in the nuclei or of such unusual character as to suggest malignant growth may be found.
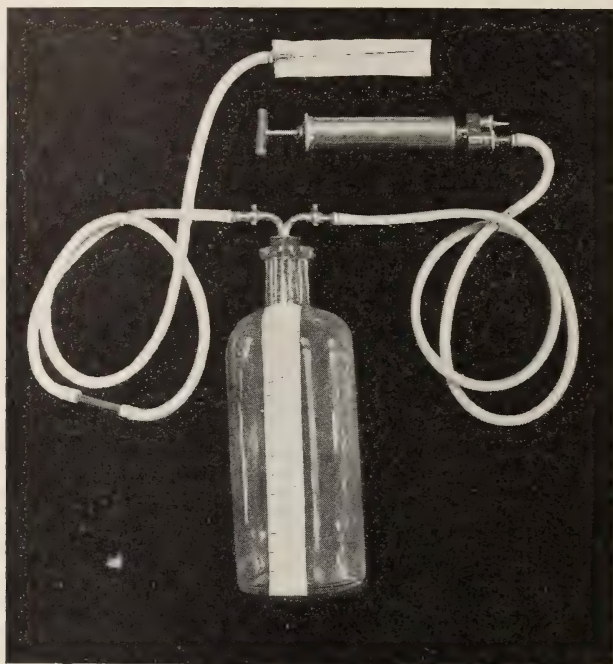
Fig. 11.—The aspiration instruments—needle, tubing, bottle and syringe.   The second tip on the syringe should be removed.   The scale on the bottle indicates the number of ounces of fluid required to reach the several levels.

The bacteriology of the fluid is also important.   The usual pyogenic bacteria may be sought in smears and cultures.   Tubercle bacilli, although present, may be demonstrable with difficulty.   If the fluid clots they should be sought in smears from the clot, or the clot may be digested and smears made from the resulting sediment (method of inoscopy).   Finally intraperitoneal inoculation of guinea-pigs with doses of from 10 to 50 cc. of the pleural fluid is most conclusive, but requires from four to six weeks for a decision.

Treatment.—In the early stages the treatment is that of dry pleurisy. With the appearance of fluid the patient should be put to bed, and a dry diet prescribed.   Efforts to control the effusion by the administration of

saline cathartics, by sweating, and counterirritation by mustard, turpentine or iodine are regularly made, but are rarely of value. The salt-free diet may be tried. Careful feeding and nursing are most important. Aspiration of the fluid must be practiced (1) if dyspnea develops from the presence of the fluid; (2) if the effusion rises above the mid-scapula, whether or no dyspnea be present; (3) if after a week or ten days the fluid shows no signs of absorption; (4) if, after removal, the fluid returns, aspiration is to be repeated as often as necessary. (See Figs. 11 and 12.)

Technique of Paracentesis.—The operation may be done with the patient lying upon one side or, better, sitting upright. The principles of asepsis must be observed as to the preparation of the patient's back and the operator's hands. The instruments to be used, commonly the
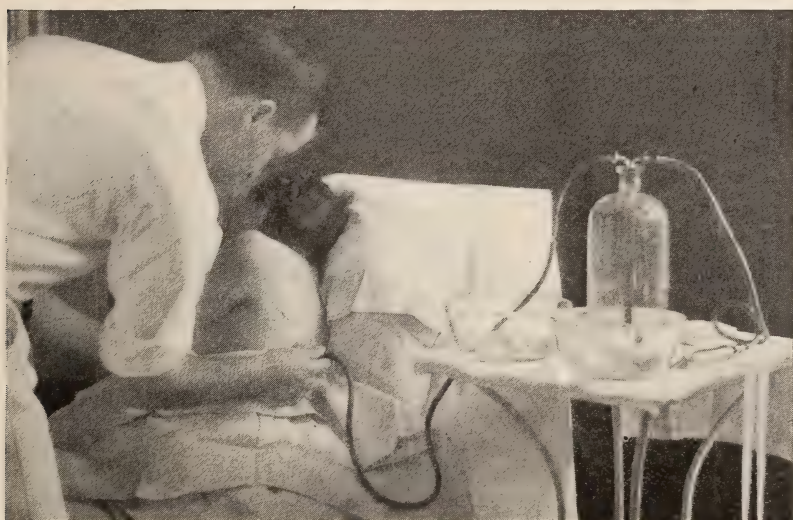
Fig. 12.—Aspiration of the chest. The curved line on the patient's back marks the position of the scapula. The needle is inserted in the 8th intercostal space.

Dieulafoy or Potain aspirator, should be sterilized. The apparatus should then be tested to see that it is working properly, and in its use the fact should never be forgotten that it is quite possible with the apparatus as made to charge the bottle with compressed air instead of evacuating it. The site of aspiration must depend upon the location of the signs of fluid: usually the seventh or eighth intercostal space below the angle of the scapula is selected. The puncture may also be made in the sixth space in mid-axilla. The fluid should be allowed to flow off slowly, and the aspiration promptly stopped if the patient shows signs of dyspnea or distress from the removal. In dealing with large effusions it is wisest not to draw off more than one litre at a time, though some clinicians advocate the removal of all the fluid possible at any time. The operation is rarely attended by bad results. Severe pain and some bleeding is regularly

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caused by contact of the lung with the sharp needle, as the fluid subsides. Hemoptysis is not uncommon. Pneumothorax often results from puncture of the lung. Subcutaneous emphysema may also develop. Severe dyspnea, edema of the lungs, sometimes accompanied by an albuminous expectoration, may result. Sudden death has occurred in some cases. The advantages of the operation far outweigh its dangers. It should be used and repeated as necessary. A preliminary injection of morphine gr. $\frac{1}{4}$ and atropine gr. 1–100 appears to prevent the distress and possibly collapse which sometimes follow the operation.

In convalescence every effort should be made to restore the vigor of the patient, and in many cases the patients should be treated as for incipient tuberculosis.

PURULENT PLEURISY
(Empyema)

Etiology.—The general etiology of pleurisy has already been given. The purulent exudates naturally occur most often as complications of processes already septic: (1) infected wounds; (2) abscesses or suppurative processes extending from the lung, the thorax, pericardium, peritoneum or other adjacent structures; (3) acute infectious diseases, most of all lobar pneumonia. The pleural effusions complicating or following lobar pneumonia are nearly always purulent, and the majority of cases of empyema are seen in that relation. It may follow other infectious diseases, especially scarlet fever or typhoid. (4) Any pleurisy with effusion may develop into an empyema. Such transformation may rarely be attributed to infection due to aspiration. In most cases it is a natural evolution.

Morbid Anatomy.—The fluid in empyema may vary from slightly turbid serum to thick pus. The odor is sometimes very disagreeable. The pleura is thickened and covered with a dense layer of fibrin. Adhesions are common and the effusions are frequently encysted. The lung is compressed and the conditions are as in pleurisy with effusion, with possibly greater displacement of the adjacent viscera. The primary focus may be found in the lungs, the thorax, or some of the adjacent viscera.

Bacteriology.—Empyema is most often caused by the streptococcus. The pneumococcus is not uncommon, and the staphylococcus is occasionally found. Tubercular empyema is sterile to cultural methods.

Symptoms.—The manner of onset and symptoms cannot be distinguished from those of pleurisy with effusion, except that when suppuration is well established and the amount of pus large, the temperature is usually higher, more remittent in type, accompanied by more profuse sweating and more rapidly developing anemia and emaciation. In severe cases there may be repeated chills, profound sepsis, and rapid exhaustion.

PHYSICAL SIGNS.—All the signs enumerated under pleurisy with effusion are found in this condition. Certain signs are usually more marked. (1) The enlargement of the affected chest is greater. (2) The displace-

ment of the heart is more marked. (3) The whispered voice, sometimes heard over clear fluid, is not transmitted (Bacelli's sign). (4) In very rare cases affecting the left side a pulsation synchronous with the heart beats is detected (pulsating pleurisy). (5) The chest walls may become edematous. (6) In rare instances an empyema may rupture through the chest walls, leaving a discharging sinus.

Course.—Empyema, unless relieved by operation, regularly results fatally. Spontaneous cure is possible, either by rupture of the pus into a bronchus and discharge of it through the mouth or through breaking down of the chest wall (empyema necessitatis, see Fig. 13). In either event recovery is long delayed and the outcome doubtful. The possibility

Fig. 13.—An empyema of the left pleura presenting externally empyema necessitatis.

of the absorption of encysted collections of pus in the pleura must be admitted. In the great majority of cases the conditions become steadily worse until the patient is relieved by operation or dies.

Prognosis.—Pneumococcus infections are mildest and as a rule run a favorable course. Streptococcus infections are likely to be most acute and most severe. The tubercle bacillus usually produces an insidious infection which has a strong tendency to become chronic, is very difficult to overcome, and is likely to be complicated by pulmonary or other tuberculosis.

Diagnosis.—The differential diagnosis is essentially that of pleurisy with effusion, except that in empyema there is usually a leukocytosis and an increase in the polynuclear percentage. This brings the clinical picture into close relation to that of pneumonia. The differentiation should

always be finally determined by the exploring needle. No other test is dependable.

Treatment.—In the early stages these cases must be treated like fibrinous or serous pleurisy. When the presence of pus is determined, drainage by surgical procedure is essential. In young children simple incision in an intercostal space and the insertion of a drainage tube are sometimes sufficient, but in most cases the resection of a rib is necessary to secure adequate drainage. In very chronic cases the resection of a number of ribs may be necessary. Cases of recovery after aspiration of an empyema are known. Small effusions, in children, produced by pneumococci may possibly yield to this mild measure, but it is not reliable.

Recently empyema has been successfully treated by repeated aspiration followed by the injection into the pleural cavity of several ounces of two per cent. formalin in glycerin. The value of the procedure has not yet been decided.

SPECIAL FORMS OF PLEURISY

Diaphragmatic Pleurisy.—Pleurisy may affect or be limited to the portion covering the diaphragm. The effusion may be of any character, fibrinous, serous, or purulent. Two features of the process are important: (1) Pain produced by diaphragmatic pleurisy is frequently referred to the abdomen and associated with rigidity, especially on the right side, has given rise to suspicion of and led to operation for inflammation of the appendix or gall-bladder. (2) Physical signs may be lacking or so slight as to be overlooked. Thoracic respiration, absence of Litten's diaphragm shadow, tenderness over the phrenic nerve in the neck, with cough and dyspnea not otherwise explained, are suggestive. (3) The symptoms other than the location of the pain are those of any similar pleurisy. Obstinate hiccough has, in some cases, resulted from the diaphragmatic pleurisy.

Sacculated Empyema.—Serous pleural exudates are rarely encapsulated, while empyemata are frequently so. The purulent exudate may be localized at any part of the chest, even the apex. A curious and rare form is that in which the pus is found encapsulated in an interlobar fissure, the interlobular empyema. Such localization gives rise to unusual localization of the physical signs. The diagnosis must be based on the constitutional symptoms taken with the localization of the signs, the results of an X-ray examination and finally the exploratory puncture. To be successful puncture must be made very deeply, and as the procedure must always be attended with some danger, exploratory incision is sometimes preferable.

Hemorrhaghic Effusions and Hemothorax.—Slight admixture of blood with pleural fluids is commonly the result of trauma by the needle. Blood may, however, be present in both exudates and transudates of the pleura.

Hemorrhagic pleurisy results (1) from tuberculosis, (2) from cancer,

(3) in rare instances from unknown cause. Occasionally after pneumonia or in purpuric conditions, or in pleurisy complicating nephritis, cirrhosis of the liver, or heart disease, the fluid may be bloody, but it rarely continues so, unless tuberculosis or cancer underlies the process. Hemorrhagic transudates are met with at times in cases of nephritis and heart disease, or possibly from obstruction of thoracic veins.

Hemothorax results from the rupture of aneurisms or the erosion of intrathoracic vessels, or trauma of the lung or chest wall. Fracture of the ribs is especially likely to cause hemothorax by puncture of the lung.

The symptoms are usually increasing dyspnea and evidences of hemorrhage. The physical signs are those of hydrothorax. The exploring needle withdraws blood. After days or weeks the blood may become infected and empyema result.

Treatment.—This is usually limited to the aseptic treatment of external wounds and the management of the general condition. The blood in the pleura is left, unless severe dyspnea is caused. It may then be aspirated, the blood being drawn off very slowly. The development of suppuration will necessitate drainage.

CHRONIC PLEURISY

Any form of pleurisy may become chronic.

Chronic Dry Pleurisy.—After any acute inflammation the pleura is left thickened and probably more or less adherent. In some instances the affected pleura becomes the seat of chronic inflammation with exacerbations of acute pleurisy from time to time, resulting in great thickening and general adhesions. The process is regularly complicated by interstitial pneumonia, chronic bronchitis, and possibly bronchiectasis. Apart from an occasional stitch in the side the symptoms are limited to those of the associated conditions. The physical signs include retraction of the affected part of the lung with depression of the chest, limited expansion, dulness on percussion, and diminished voice and breathing sounds. (See Interstitial Pneumonia.) At the base such signs indicate, as a rule, a simple process; at an apex they regularly signify tuberculosis. In the postmortem room more or less general thickening of the pleura with adhesions, not recognized during life, is frequently found.

Chronic Pleurisy with Effusion.—Cases of serous pleurisy or empyema may be so protracted as to be termed chronic. In such cases the pleura becomes greatly thickened, and the lung is compressed and rendered useless. The patients give the usual symptoms and signs of pleurisy and become emaciated and feeble as the result of long illness.

Treatment.—The general health of the patient must be the chief object of attention. In the dry form gymnastic and breathing exercises are employed to expand the affected part of the lung and prevent retraction. In the serous form, after repeated tapping, drainage as for empyema may be required. In empyema Estlander's operation to secure the obliteration of the pleural cavity may be called for.

HYDROTHORAX

Definition.—An accumulation of serous fluid not of inflammatory origin in the pleura—a transudation.

Etiology.—The condition is secondary to (1) cardiac or renal disease, (2) anemia or other hydremic condition of the blood, or (3) the pressure of tumors or new growths upon some of the thoracic veins. The symptoms are those of the underlying condition with a gradually increasing dyspnea, due to the accumulating fluid. There is no pain or fever. The signs are those of pleurisy with effusion, except that the heart is less likely to be displaced. In cardiac hydrothorax the effusion is most often on the right side. The pleural fluid is clear serum, alkaline to litmus, with a specific gravity of 1.010 to 1.015, much albumin, and as a rule an excess of endothelial cells.

Treatment.—The underlying condition must be treated. With severe dyspnea the fluid may be aspirated, but will quickly reaccumulate, unless the primary affection is controlled.

PNEUMOTHORAX, HYDROPNEUMOTHORAX, AND PYOPNEUMOTHORAX

Pure pneumothorax is a very rare condition, but collections of serum or pus with air or gas in the pleura are not unusual.

Etiology.—Air or gas may be admitted to the pleura from three sources. (1) Through wounds of chest wall or lesions of the lung air may enter the pleura. (2) By rupture gas in the stomach or intestine may enter the pleura. (3) Finally, infection of pleural exudates by certain bacteria, especially the bacillus aërogenes capsulatus, may result in the production of gas. The great majority of cases belong in the first category. (a) Wounds of the chest wall or lung may admit air at any time. Punctures made by the exploring or aspirating needle may do so, although these instruments are more likely to wound the lung. (b) A normal lung may rupture under unusual strain. (c) A diseased lung is much more liable to such an accident. From 70 to 90 per cent. of all cases of pneumothorax are due to the rupture of a tuberculous focus in the lung. Bronchopneumonic foci may also yield under strain, especially in children suffering from whooping-cough. The two other origins of pneumothorax are very rarely seen. When the chest wall or lung is penetrated air enters the pleura by reason of the elasticity of the lung which leads it to retract and creates a condition of negative pressure in the pleura which is met by the entrance of air, until an equilibrium is established. By reason of the same tendency on the part of the other lung the entrance of air may continue till the affected pleura is somewhat distended, the heart crowded to the opposite side and the diaphragm depressed. In consequence of a valve-like action of the opening in the lung this distention of the affected side may go on to extreme displacement of the heart and other adjacent viscera.

Morbid Anatomy.—The lung on the affected side is completely re-tracted and lies as a shrunken mass in the upper and posterior part of the chest. The pleura may be empty, but regularly contains either serum or pus. The pleura itself is congested and often coated thickly with fibrin, especially in the purulent cases. In the great majority of cases the lung presents tuberculous lesions. It may be difficult to locate the rupture. The heart is usually displaced to the other side, the diaphragm, liver, and spleen depressed.

Symptoms.—These depend greatly upon the amount of air drawn into the pleura and the resulting displacement of the heart and other organs. The onset is usually sudden. Sharp pain in the chest may be felt at the time. Dyspnea succeeds, at times so slight that the patient continues his occupation, at other times severe. In the milder cases there are no other symptoms, in the severe the dyspnea is intense, the face and body become livid, pulse rapid and feeble, the extremities cold, the body bathed in cold sweat, and the patient is in danger of immediate death.

Course and Prognosis.—If the patient survives the initial shock, the outcome depends upon the nature of the lesion. In the spontaneous cases, or in those produced by wounds of an aspirating needle, the air is usually absorbed and the patients are soon well. Pneumothorax resulting from accidental puncture of the lung in aspiration may prove a dangerous or possibly fatal complication of pleurisy with effusion or hydrothorax. The tuberculous cases, the great majority, do badly. The pleura becomes infected, the patients suffer from pyopneumothorax as well as tuber-culosis, and gradually failing die within a few weeks or months.

Physical Signs.—Inspection.—The affected side may bulge and is immobile. The pulsation of the displaced heart may be noted. Cyanosis, dyspnea, and profound exhaustion may be present.

Palpation.—The heart beat is located in an unusual position. (See Fig. 14.) Vocal fremitus is absent over the air-containing chest, also over the area of fluid, if this be present. The liver or spleen is depressed so as to be palpable.

Percussion.—Over the air-filled chest, the note is tympanitic, hyper-resonant, or dull. The area of resonance is larger than normal, if the pleura is distended. If fluid be present, there is dulness over it, shifting as the position of the patient is changed. The heart dulness is displaced or lost.

Auscultation yields the most characteristic signs of pneumothorax. The respiratory murmur is faint and may be lost over the air, but is frequently amphoric. It will be lost over fluid. The voice sounds are similarly faint and distant, but may be amphoric. Moist râles may be heard and the metallic tinkle of Laennec. The coin test gives positive results. Thus the clink produced by holding one coin on the chest in front and tapping it lightly with another is heard clearly if one listens over the posterior chest. On shaking the patient a succussion or splashing sound is heard if fluid is present with the air. The metallic tinkle, the coin test and succussion are practically pathognomonic of hydropneu-mothorax.

Diagnosis.—With characteristic physical signs the diagnosis of pneumothorax is easy. Pleurisy with effusion may give similar signs, but without the metallic tinkle, coin test, or succussion. Dilated stomach may sometimes give such signs in the lower left chest, but the tympany is continuous with that of the stomach in the abdomen with the usual evidences of dilatation of the stomach. Diaphragmatic hernia admitting the stomach and intestines to the thorax may easily be mistaken for hydropneumothorax. Variations in the signs, the presence of borborygmi and the results of fluoroscopy after a bismuth meal will identify the condition. Subphrenic abscess containing gas may present a very difficult problem. Here a previous history of gastric or intestinal ulcera-
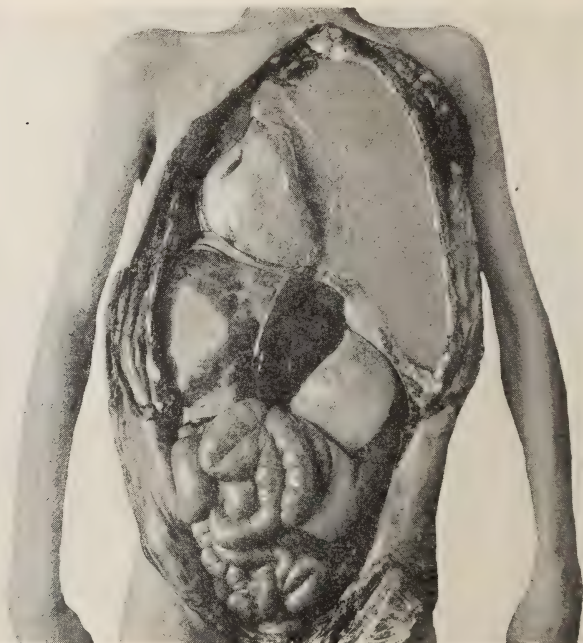
Fig. 14.—Pyopneumothorax: left pleura filled by pus (and air) concealing the lung; the heart pushed far to right and covering all but the anterior edge of the lung; the left side of diaphragm, left lobe of liver, stomach, spleen and colon pushed downward and to right. From the collection of Dr. Walter B. James.

tion, absence of pulmonary symptoms, the normal situation of the heart, and epigastric pain and tenderness should lead to a correct diagnosis.

Treatment.—Rest in bed and good care suffice for the simpler cases. If much fluid be present, or if the displacement due to air be extreme, aspiration is indicated and may be repeated as often as necessary. Operative treatment, other than aspiration, is rarely helpful, since the wound in the lung fails to close, the pleura, if not already infected, becomes so and the patient dies of protracted suppuration. For the great majority the after-treatment is that of pulmonary tuberculosis.

PLATE I.

LESIONS OF THE MOUTH.

A, Parasitic stomatitis.   B, Ulcerative stomatitis.   C, Aphthous stomatitis.
                          D, Geographical tongue.
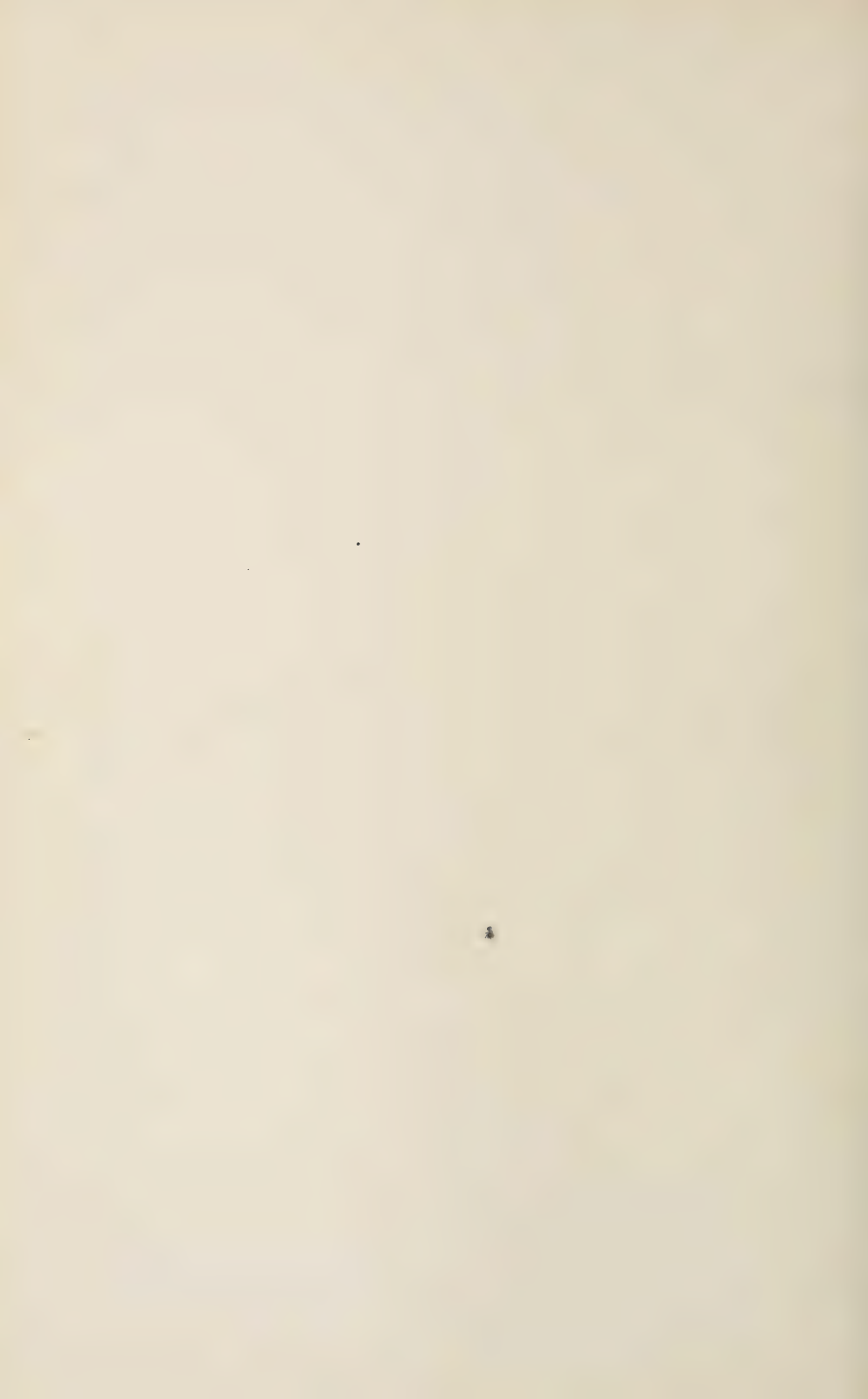
DISEASES OF THE DIGESTIVE SYSTEM

DISEASES OF THE MOUTH

STOMATITIS

Inflammation of the mucous membrane of the mouth occurs in various forms and under many different conditions. It is seen in such general diseases as scarlet fever, diphtheria, scurvy, etc.; in various digestive disturbances; as a result of local irritation or infection of various sorts, and after the administration of such drugs as mercury.

Catarrhal Stomatitis.—Simple inflammation of the buccal mucosa is seen at all ages and is caused by various local irritants, by dentition, by digestive disturbances, etc. It is often seen in the infectious diseases. Usually a large portion of the mucosa of the mouth and tongue is involved and is bright red, and swollen, tender and painful. Constitutional symptoms are slight or lacking. TREATMENT consists in the careful cleansing of the mouth after feeding and in the frequent use of a simple mouth wash such as lime water, or a solution of sodium bicarbonate or borax (gr. x–ʒi).

Herpetic Stomatitis (Aphthous Stomatitis).—This is a condition common in young children and marked by the appearance of small herpetic vesicles upon the edges of the tongue and inner surface of the lips and cheeks. Within a day or two the vesicles break and superficial ulcers with a grayish base and red periphery are formed. There are usually several crops of such vesicles and the condition lasts a week or two. (See Plate I.) There is pain and burning in the mouth, increased salivation and difficulty in taking food. There may be slight fever. The mouth should be frequently cleansed with some simple mouth wash and the vesicles or ulcers touched with nitrate of silver or powdered burnt alum.

Ulcerative Stomatitis.—This is a more severe form of inflammation which may occur at any age, but which is chiefly seen in children. Most of the cases are met with among children of the lower classes, and malnutrition from any cause, and a lack of cleanliness of the teeth and mouth are the chief predisposing factors. The same condition is seen in scurvy and in mercurial poisoning. The gums become swollen and spongy and there appear at the edges of the gums and between the teeth small, irregular ulcers having a dirty gray base and ragged edges. In severe cases the ulcers may extend to the inner surface of the cheeks and to the tongue; the teeth may loosen and there may even be necrosis of the alveolar process. (See Plate I.)

Profuse salivation and fetid breath are the most noticeable symptoms. The gums bleed readily; there is great pain and discomfort in taking food; the submaxillary lymph-glands are swollen, and there is usually some fever. The disease, unless properly treated, may last many weeks and may even threaten life.

TREATMENT.—Everything possible should be done to improve the general health and to remove the primary cause. The mouth should be frequently washed with peroxide of hydrogen, diluted two to five times, or a weak solution of potassium chlorate or of alum. Internally potassium chlorate acts as a specific and should be given in small and frequently repeated doses (gr. ii–v). Under such treatment recovery is usually prompt and complete.

Parasitic Stomatitis (Thrush).—Thrush occurs chiefly in infants during the first few months of life. It is due to infection by a fungus, *saccharomyces albicans,* which gains entrance to the mouth by means of nursing bottles, soiled nipples, etc., or through the air. Some slight inflammation of the mucosa or lack of cleanliness seems necessary for infection. The fungus is composed of branching threads (mycelium) and spores. It invades the epithelial layer of the mucosa and forms small patches of milk-white color which unite to form large areas. The process is seen chiefly upon the dorsum of the tongue, sides of the cheeks and hard palate, but may extend to the wall of the pharynx and even down the esophagus to the stomach. This white pellicle, which looks like coagulated milk, is quite firmly attached to the mucous membrane and when removed is apt to leave bleeding points. (See Plate I.) The only *symptoms* are those of some discomfort in taking food. The condition is of much importance in poorly nourished and sickly infants in whom the disinclination to nurse may be a serious matter.

TREATMENT.—The disease is much more easily prevented than cured. If nursing bottles, rubber nipples, etc., are properly disinfected, and the mouth cleansed after each feeding, thrush is not likely to develop. When it exists a saturated solution of boric acid or some other mild antiseptic should be applied several times a day and the mouth should be cleansed after each feeding with a solution of borax or of sodium bicarbonate (gr. x–ʒi).

Gangrenous Stomatitis (Cancrum Oris, Noma).—This is a rare and fatal affection occurring in children and characterized by a rapidly spreading gangrene of the mouth and cheeks. The disease develops in children whose vitality and resisting powers have been much lowered by some illness, and is usually seen after some such infectious disease as measles. The *Streptococcus pyogenes* or an organism resembling closely the diphtheria bacillus is usually found. The process begins in the mucous membrane of the cheek or gums as a small spot of gangrene which extends rapidly until a large portion of one or both cheeks is destroyed. The surrounding tissues are edematous and inflamed. The process usually shows no tendency to self-limitation, but occasionally the slough may separate and the remaining ulcer cicatrize and heal. The

symptoms are those of profound sepsis. There are high fever, extreme prostration and a sickening fetor to the breath. Most of the cases die within a week or ten days.

Treatment is altogether unavailing unless the disease is recognized and radically treated by excision and cauterization in its earliest stage. The patient's strength should be supported by careful feeding and free stimulation.

Acute Glossitis.—An acute inflammation of the tongue is occasionally seen as the result of trauma, of the taking of too hot food or drink or of some corrosive substance. It also occurs in mercurial stomatitis. The tongue is much swollen, red, and indented by the teeth. Salivation is increased and there are great pain in taking food and difficulty in swallowing. The condition usually subsides within a few days and no treatment is needed except a simple astringent mouth wash.

Geographical Tongue.—This is a sub-acute or chronic affection of unknown origin, seen occasionally both in children and in adults. Upon the dorsum of the tongue there appear bright red areas of circular or crescentic outline with gray borders, which gradually spread, coalesce and change their outline. The red areas are due to desquamation of the superficial epithelium, the gray borders to thickening of the epithelium. (See Plate I.) Aside from some slight sensitiveness of the tongue there are no symptoms. The condition subsides within a few weeks or months.

Leukoplakia Buccalis.—This chronic affection of the tongue is seen most frequently in heavy smokers. A similar condition is met with in syphilis. Slightly elevated, irregular whitish patches appear upon the dorsum, due to thickening of the superficial epithelium, which run a very chronic course, cause more or less discomfort in eating and are very resistant to treatment. Rarely the condition has terminated in epithelioma.

DISEASES OF THE SALIVARY GLANDS

Disturbances of the Salivary Secretion.—Excessive secretion of saliva (*Salivation*, *Ptyalism*) may result from the use of such drugs as mercury, pilocarpine, tobacco and potassium iodide; is often seen in pregnancy and insanity, and occurs in inflammations of the mouth and in hydrophobia.

A lack of salivary secretion (xerostomia) is seen in fevers, in certain stomach disorders, in diabetes and in poisoning by belladonna and stramonium.

Inflammation of the Salivary Glands.—The parotid is the gland chiefly affected.

ACUTE PAROTITIS.—Aside from the specific form (mumps) parotitis occurs as a *secondary* affection in typhoid fever, pneumonia and many other infectious fevers, and occasionally in injuries and diseases of the genital organs and abdominal viscera. Infection may occur from the mouth along Stenson's duct, or by means of the blood. In many of these

cases the iflammation goes on to suppuration and pus may be expressed from the duct. Usually the process subsides within a week or ten days.

Chronic Parotitis may result from the acute form or from poisoning by mercury or lead.

Von Mikulicz's Disease is a rare and curious affection characterized by the slow and symmetrical enlargement of all the salivary, and of the lachrymal, glands. Its cause is unknown. The parotid gland may be the seat of both benign and malignant tumors.

DISEASES OF THE PHARYNX

PHARYNGITIS

Acute Catarrhal Pharyngitis.—This may result from exposure alone, but is favored by such conditions as gout, rheumatism and digestive disturbances. The inflammation may be confined to the pharynx or may involve also the tonsils, soft palate, Eustachian tubes, larynx, etc. Constitutional disturbance is usually slight, but there are pain, dryness and tickling in the throat, discomfort in swallowing and often soreness of the muscles of the neck. The trouble usually subsides within three or four days.

A brisk purge at the outset, salol or aconite in small doses and a simple throat spray are all that is needed in the way of treatment.

Chronic Pharyngitis.—Chronic inflammation of the pharynx is frequently seen in heavy smokers, in clergymen, public speakers and others who use the voice excessively and in those of a gouty or lithemic diathesis. The mucous membrane is congested and covered with thick, tenacious mucus. In some cases it is much thickened; in others, it is thin, dry and shiny (*pharyngitis sicca*), and in others many minute, projecting red areas are seen (*granular pharyngitis*). The small projections are composed of masses of lymphoid cells. The symptoms are those of dryness and tickling in the throat with the constant inclination to cough and clear the throat. Sometimes the constant accumulation of mucus causes much discomfort. Hoarseness and cough are often the result of an associated laryngitis. In the treatment attention must be paid to the removal of the cause and the improvement of the general health. Occasional applications of a solution of nitrate of silver (10 per cent.) should be made to the pharynx and the throat should be kept free of mucus by the frequent use of a simple spray.

Retro-pharyngeal Abscess.—Two distinct varieties occur: (1) an acute form seen in infants and (2) a chronic form secondary to caries of the cervical vertebræ.

The acute form is seen in children of less than three years and begins as an infection of the retro-pharyngeal lymph-glands, which does not always go on to suppuration. The general symptoms are those of fever and prostration and the local symptoms those of nasal and laryngeal obstruction and difficulty in swallowing. There are nasal voice, mouth

breathing, dyspnea which is chiefly inspiratory, and difficult and painful deglutition. Inspection of the pharynx and examination with the finger reveal a fluctuating tumor mass projecting forward from the posterior pharyngeal wall. The swelling may show externally below the angle of the jaw and there may be stiffness of the neck. The diagnosis offers no difficulties if the possibility of the condition is borne in mind, and the throat examined by the eye and finger.

Treatment consists in the prompt evacuation of the abscess by incision through the mouth.

THE CHRONIC FORM is much rarer, occurs at any age, is usually much larger and is associated with little or no fever. The spine gives evidences of caries.

DISEASES OF THE TONSILS

ACUTE FOLLICULAR TONSILLITIS

Etiology.—This is an extremely common affection seen chiefly in older children and young adults. Exposure to cold and damp, unhygienic surroundings, tonsillar hypertrophy and rheumatism seem to be predisposing causes. Individual predisposition varies greatly. The disease often attacks several members of a household.

Morbid Anatomy.—The inflammation involves the crypts and surface of the tonsils, which are red and somewhat swollen. At the mouth of the crypts small yellowish-white plugs appear, which consist of epithelial and pus cells, mucus and fibrin. Both tonsils are affected.

Symptoms.—The onset is abrupt with chilliness, headache, pains in the back and limbs and a rapid rise of temperature to 102°–104°. The tongue is coated and there are anorexia and constipation. The general symptoms are usually quite out of proportion to the local ones, which consist only of soreness of the throat and discomfort in swallowing. The surface of the tonsil is studded with yellowish-white points, some of which may fuse together, but there is no true pseudo-membrane and the points can be readily wiped away with a swab. Most of the symptoms subside in three or four days and the whole duration of the disease is less than a week. Otitis media is an occasional, and endocarditis a rare complication. The differential diagnosis from diphtheria is given under that heading.

Treatment.—Rest in bed, a fluid or semi-fluid diet and an active purge at the outset are the chief requisites. Small doses of aspirin or sodium salicylate are given at frequent intervals. The pains may be relieved by phenacetin. The throat should be sprayed with some simple alkaline solution or with dilute hydrogen peroxide. A cold compress may be applied to the neck, or hot applications such as a poultice of flaxseed or kaolin (see Appendix) may give comfort.

SUPPURATIVE TONSILLITIS
(Quinsy)

The etiology is similar to that of follicular tonsillitis.

The inflammation begins deep in the substance of the tonsil or in the peritonsillar tissue and in most cases goes on to the formation of an abscess. Usually only one tonsil is attacked.

Symptoms.—The onset does not differ from that of follicular tonsillitis. Soon, however, the local symptoms become much more pronounced. There is constant severe pain in the throat, great difficulty in swallowing, stiffness and tenderness of the neck and difficulty in opening the mouth. One tonsil and the surrounding tissues then become greatly swollen and may extend to or beyond the median line, the uvula being displaced to the opposite side. At first the swelling is firm and brawny, but after several days an area of softening can usually be recognized on the anterior surface. The abscess if left to itself breaks in from five to seven days, and this is usually followed by prompt relief of all the symptoms, both general and local, and by rapid convalescence. The patients regularly recover. Rarely death has occurred from edema of the glottis or from perforation of the carotid artery. Occasionally the process is repeated in the other tonsil.

Treatment.—In addition to that given for follicular tonsillitis, the pain and distress may be somewhat mitigated by hot fomentations or an ice-bag to the neck, and by codeine or Dover's powder at night. As soon as any point of softening can be felt by the finger the tonsil should be freely incised and the pus evacuated.

HYPERTROPHY OF THE TONSILS AND ADENOID VEGETATIONS

Chronic enlargement of the tonsils and adenoid growths of the naso-pharynx are usually found associated and produce similar symptoms.

Etiology.—These conditions occur chiefly in children up to the time of puberty. A marked predisposition to lymphoid hyperplasia is seen in certain families and individuals. Damp dwellings, bad air and frequent acute inflammations of the nose and throat are the predisposing causes.

Morbid Anatomy.—The adenoid growths are similar in structure to the tonsils and in both there is hyperplasia of the lymphoid tissue and the connective-tissue stroma in varying proportions. The tonsils are much enlarged and the adenoid growths may almost completely fill the naso-pharynx.

Symptoms.—There is a tendency to frequent attacks of tonsillitis and of nasal and pharyngeal catarrh. Obstruction of the posterior nares by the adenoid growths results in mouth-breathing, snoring, restless sleep, night terrors, a hoarse, nasal voice, deafness, a dull, apathetic facial expression, a high, narrow palatine arch and certain deformities

of the chest (pigeon breast). The breath is often very offensive and there may be various reflex symptoms. Acute otitis media is a frequent sequel. The diagnosis of adenoid growths is verified by digital examination of the naso-pharynx.

Treatment.—The milder grades of tonsillar and adenoid enlargement may be somewhat relieved by general tonic treatment, by the use of the syrup of the iodide of iron (gtt. x–xx t. i. d.) and especially by removal to a warm, dry climate during the winter months. When the symptoms are pronounced, however, the operative removal of both the adenoids and the tonsils is demanded, since very serious and permanent damage to the child may thus be prevented.

DISEASES OF THE ESOPHAGUS

ACUTE ESOPHAGITIS

This is most frequently seen as the result of the swallowing of corrosive poison or of very hot fluids. It is seen occasionally in the course of such diseases as typhoid fever, pneumonia and small-pox. A phlegmonous type sometimes complicates ulcer or cancer of the esophagus. The inflammation may be of a catarrhal, suppurative or pseudo-membranous type.

The chief *symptom* is a constant, dull or burning pain behind the sternum which is much increased by every effort to swallow food. There is usually more or less fever. If ulceration develop the resulting cicatrization usually leads to stricture.

ULCERATION OF THE ESOPHAGUS

Ulceration is rare except as a result of corrosive poisons or of cancer. It may occur, however, in typhoid fever, small-pox, diphtheria, syphilis, and tuberculosis. An ulcer similar in nature to the peptic ulcer of the stomach is sometimes seen at the lower end.

Varicosities of the esophageal veins often occur in cirrhosis of the liver with portal obstruction, and profuse and even fatal hemorrhage may result from their rupture.

SPASM OF THE ESOPHAGUS

Aside from hydrophobia this rare condition is met with chiefly in hysterical or hypochondriacal patients of either sex. There is a sudden contraction of the muscular coat which prevents the swallowing of solid food or even of liquids. It is paroxysmal in its appearance; occurs especially when the patient is excited or nervous, and is often associated with thoracic pain. The nutrition usually does not suffer. The condition is recognized by the history and by the fact that the spasm will yield to the steady, gentle pressure of an esophageal bougie. The treatment is that of the underlying nervous state.

STENOSIS OF THE ESOPHAGUS

In addition to the spasmodic stricture just described, obstruction of the esophagus may result from (a) cicatricial contraction; (b) cancer or other new growth in the wall; (c) pressure from without, by thoracic aneurism, mediastinal tumor, pericarditis with effusion, goitre, etc., and (d) foreign bodies.
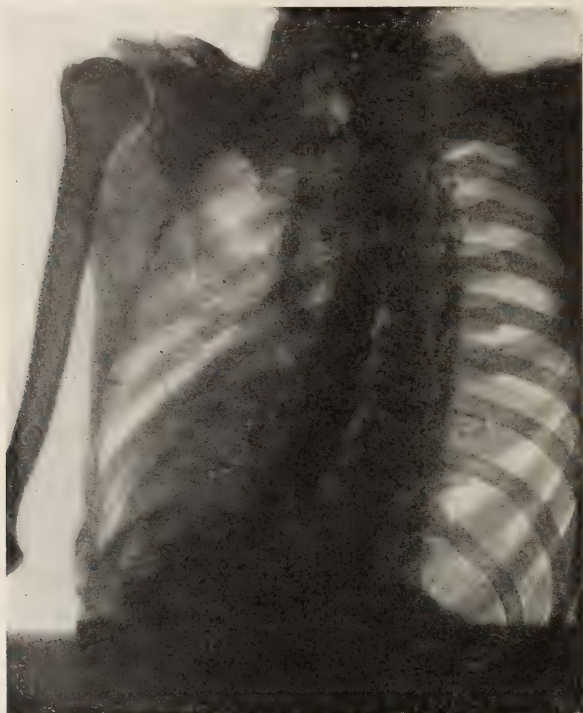
FIG. 15.—Stricture and dilatation of the esophagus, demonstrated by the X-ray after the ingestion of an emulsion of bismuth subcarbonate, which being impervious to its rays appears as a dark shadow. The patient lies obliquely upon the right side and the X-ray tube is close to the left shoulder, which is therefore most distinct. The curvature of the spine results from the position.

Cicatricial Stricture.—The extensive ulceration which follows the swallowing of some corrosive poison is the usual cause. The stricture is commonly near one or the other end of the esophagus, and the tube above is apt to be dilated and its muscular wall hypertrophied. There is increasing difficulty in the swallowing of solid food. If there is much dilatation above the stricture the food may be retained for some time before it is regurgitated. Such regurgitated material will be undigested and free from hydrochloric acid and from the characteristic odor of stomach contents. The position and calibre of the stricture may be recognized by the careful passage of graduated esophageal bougies. The treatment is surgical.

Cancer of the Esophagus.—This occurs usually in advanced life and is commoner in men. The growth begins in the mucous membrane, is of the type of epithelioma and is commonly seen in the lower third of the tube. From its annular form it tends to early and progressive stricture, which, however, is sometimes temporarily relieved by the ulceration to which the tumor is prone. This ulceration may perforate the wall and cause infection of the cellular tissue of the neck, the posterior mediastinum, the pericardium, lung or trachea, etc.

The SYMPTOMS are those of increasing difficulty in swallowing solid, and later liquid, food, together with progressive emaciation and more or less pain. The regurgitated food may be mixed with blood or pus, and the tip of the exploring sound may be blood stained. Death results from starvation or from some of the complications due to perforation.

The DIAGNOSIS is made by excluding the other above-mentioned causes of obstruction and by considering the age of the patient. The possibility of the presence of a foreign body or an aneurism should be remembered. The disease is always fatal.

TREATMENT is purely palliative. So long as liquid food is swallowed without difficulty nutrition may be fairly well preserved. Sometimes feeding may be accomplished by means of a small stomach tube passed beyond the point of stricture. When swallowing becomes inadequate gastrostomy should be performed, since rectal feeding cannot be maintained for any length of time. The pain may require morphine.

Diverticulum.—This rare condition is usually due to *pressure,* exerted during swallowing, and occurs in the posterior wall at the junction of pharynx and esophagus, where the muscular coat is deficient. It may reach large size. Small diverticula are sometimes seen on the anterior wall due to *traction* exerted by inflamed and adherent bronchial glands.

Dilatation of the esophagus is seen often above a stricture, but occurs rarely also as a primary condition without known cause. Stricture and dilatation of the esophagus can be beautifully shown by a radiograph immediately following the ingestion of a paste or gruel containing an ounce of bismuth subcarbonate. (See Fig. 15.)

DISEASES OF THE STOMACH

ACUTE GASTRITIS
(Acute Indigestion. Acute Gastric Catarrh)

Etiology.—Acute gastritis is common at all ages, but is especially frequent in children. Its prevalence is greatest during the hot months. Gout, tuberculosis, and other chronic diseases, and such conditions as produce passive congestion of the stomach (valvular disease, cirrhosis of the liver, etc.) are all predisposing causes. Individual predisposition is frequently seen. The condition not infrequently occurs in the course of the infectious fevers. Some error in diet is the usual exciting cause. This may be food, excessive in quantity or irritating in character, such as unripe or over-ripe fruit, sour milk, tainted meat, etc. Alcoholic

excess is a common cause. In certain persons special articles of diet, such as shell fish, etc., will always excite an acute gastric catarrh.

Morbid Anatomy.—The process is similar to that of an acute catarrhal inflammation of any other mucous membrane. The mucosa is swollen, hyperemic and infiltrated with serum and leukocytes. The surface, at first dry, is later covered with thick, glairy mucus. The cells of the mucous and peptic glands undergo cloudy swelling and the secretion of hydrochloric acid and pepsin is diminished or entirely abolished.

Symptoms.—Anorexia, nausea and vomiting are the chief symptoms. The first two are almost never absent. In severe cases vomiting is constant and persistent and there are moderate fever, much prostration and more or less epigastric pain and tenderness. In the mild cases vomiting may occur only at the outset or may be absent altogether, and there is usually little or no fever. The tongue is heavily coated; the breath foul; thirst is often distressing, and the bowels may be either constipated or loose. The vomited matter consists at first of undigested food and later of mucus (which may be blood-streaked) and bile. There is often an absence of free hydrochloric acid. The symptoms usually last only two or three days, but occasionally they may persist for a week or two, or even longer. Complete recovery is the rule, but repeated attacks may lead to a chronic gastritis.

Treatment.—At the outset, if vomiting has not occurred, the stomach should be emptied by the use of a simple emetic, and the bowels should be freely moved by calomel, blue mass or a saline. Complete rest to the stomach is the most essential part of the further treatment. Often it is best to withhold all food for 24 hours and then to begin with milk or broths in minute and frequently repeated doses. If the onset is violent a small dose of morphine subcutaneously will often check the vomiting. In other cases rectal feeding may be needed for two or three days. Vomiting may sometimes be checked by a mustard paste applied to the epigastrium or by dilute hydrocyanic acid ($\mathfrak{m}$ j–ij), bismuth subnitrate (gr. x–xx) or cerium oxalate (gr. v). The return to solid food should be made gradually and cautiously and its digestion is often aided by the use of dilute hydrochloric acid.

Special Types.—Toxic Gastritis.—A very intense form of inflammation is that due to the ingestion of the violent irritant poisons, such as the mineral acids, the caustic alkalies, carbolic acid, phosphorus, arsenic, corrosive sublimate, etc. There are necrosis and destruction of portions of the mucosa, inflammatory swelling of the surrounding areas, and submucous hemorrhages. There are also often excoriations of the lips, tongue, pharynx and esophagus.

The symptoms consist of intense pain in the throat and epigastrium, persistent retching and vomiting, difficulty in swallowing and prostration amounting often to collapse. The vomitus is often streaked with blood and may contain shreds of necrotic tissue or evidences of the poison. If the case is not immediately fatal, ulceration, chronic gastritis or esophageal stricture may result.

The TREATMENT consists in the prompt washing of the stomach, the use of the appropriate antidotes, the relief of pain by morphine, and rectal feeding.

PHLEGMONOUS GASTRITIS is an extremely rare affection in which suppuration, either circumscribed or diffuse, occurs in the submucous tissue. The cause is usually obscure. The vomitus may contain pus, but the condition is rarely recognized during life.

CROUPOUS OR MEMBRANOUS GASTRITIS occurs rarely in diphtheria and in such conditions as pneumonia and small-pox.

PARASITIC OR MYCOTIC GASTRITIS may be seen as the result of the growth of certain fungi.

CHRONIC GASTRITIS

(Chronic Gastric Catarrh.   Chronic Dyspepsia)

Etiology.—Chronic catarrhal inflammation of the stomach may be *primary* or may be *secondary* to one or more attacks of acute gastritis or to gastric ulcer or cancer. The primary cases are due regularly to long-continued dietetic errors. Of these the abuse of alcohol is perhaps the most frequent; but habitual overeating, hurried eating, insufficient mastication, the excessive use of fried dishes, of pastries, of hot breads, of tea and coffee and of ice-water are common and important causes.

Among the *predisposing causes* are to be mentioned such constitutional disturbances as gout, diabetes, chronic nephritis, chronic tuberculosis and anemia, and such conditions as produce passive congestion of the stomach (cirrhosis of the liver and chronic heart affections).

Morbid Anatomy.—The changes are usually most distinct in the pyloric portion. The mucous membrane is of grayish color, is coated with thick, tough mucus, and may show areas of congestion or pigmentation. It is also often thickened and wrinkled and may even present polypoid projections. Small, superficial erosions are sometimes seen. Microscopically the glands show various degrees of degeneration and atrophy and there are a round-celled infiltration and connective-tissue proliferation in the interstitial tissue between and beneath the glands and in the submucous and even the muscular layer. In certain cases this connective-tissue proliferation leads to great sclerosis and thickening of the stomach wall. This thickening may be confined to the pyloric region and may result in stenosis with secondary dilatation of the stomach, or rarely it may involve the whole stomach and cause great contraction of the organ (*cirrhosis ventriculi*). In other cases the inflammatory changes result in great thinning and atrophy of the mucous and muscular coats with almost complete obliteration of the gastric tubules (atrophic gastritis).

Symptoms.—The disease begins very insidiously and the symptoms vary greatly in different cases and are often vague and indefinite. Nausea, vomiting, a sense of weight and fullness, dull pain, eructations

of gas and sour fluid, and flatulence are the most common symptoms. Vomiting may be slight or lacking or may be of daily occurrence. In alcoholic cases it often occurs before breakfast. Pain when present is referred to the epigastrium and is usually of a dull, burning character. It may be the chief symptom and usually appears from one to three hours after eating. A moderate degree of epigastric tenderness is common. The tongue is usually coated, there is often a bitter taste in the mouth, the breath is offensive and the pharynx is usually congested and coated with mucus. The bowels are apt to be constipated, but diarrhea may result from the irritation of undigested and fermenting food in the intestine. The appetite is usually much disturbed, there is some loss of flesh, and headache and mental depression are common. Certain cases of atrophic gastritis are associated with a very severe and pernicious form of anemia.

EXAMINATION OF THE STOMACH CONTENTS.—The vomited matter is neutral or slightly acid in reaction, consists of partly digested food mixed with more or less thick, glairy mucus and may contain bile and rarely traces of blood. In the stomach washings there is almost always more or less mucus. After a test meal the expressed contents show usually a marked deficiency of both hydrochloric acid and pepsin; occasionally, however, HCl may be normal or increased in amount. In atrophic gastritis there may be complete absence of HCl and of pepsin and the milk-curdling ferment (*achylia gastrica*). If pyloric obstruction or atony of the stomach wall exists the food is delayed in the stomach, undergoes fermentation with the formation of abnormal organic acids, and the stomach washings will contain food remains even seven or eight hours after a full meal.

Course.—Chronic gastritis once thoroughly developed usually persists indefinitely. Improvement may be secured by treatment and complete recovery is possible, but in most cases the condition improves only temporarily and frequent relapses occur. The patients become thin, anemic, and possibly emaciated, suffer more or less constantly, and as a rule become extremely irritable and unhappy. The condition is rarely fatal in itself. The patients in the end succumb to intercurrent disease.

Diagnosis.—The presence of considerable amounts of mucus in the stomach washings, the diminution or absence of HCl in test analyses, the long duration of the symptoms and the absence of the usual evidences of gastric ulcer, cancer or dilatation are the points of special diagnostic value.

Treatment.—GENERAL MANAGEMENT.—Careful attention should be given to the removal of the cause if that can be discovered. The teeth should be put in good condition, the food eaten slowly and mastication performed thoroughly. A short rest both before and after eating, and a certain amount of regular exercise and recreation should be insisted upon.

DIET.—No hard and fast rules can be laid down. In severe cases it may be wise to confine the diet for a time to skimmed milk, milk and

vichy, buttermilk or koumyss. In most cases it is only necessary to have the meals light, well cooked and easily digestible, and to exclude such unsuitable articles as hot breads and cakes, pastries, fried foods, fats, pork, veal, etc. Sugar and starches should be used sparingly if there is much fermentation and flatulence. Alcohol, coffee and tea should be taken only in very small quantities if at all.

LAVAGE.—In most cases lavage is of great value. It is especially useful in cases in which mucus is abundant or in which there is a tendency to the retention of food beyond the normal period. The water should be warm and should be made alkaline by sodium bicarbonate ($\frac{2}{3}$i to the quart). Weak solutions (1–2000) of nitrate of silver are also used. The washing is best done at bedtime or before breakfast.

MEDICINES.—Hydrochloric acid is useful whenever its secretion is deficient. Twenty or thirty minims of the dilute acid should be given after meals. Pepsin is rarely lacking in the gastric juice and is therefore not often required. Sodium bicarbonate and the bitter tonics given before meals are often an effective stimulant to gastric secretion.

Pain may usually be relieved by bismuth subnitrate or by small doses of cocaine. Constipation is to be combated by a laxative diet (cooked fruits, buttermilk, honey, etc.) and by a simple aloin or cascara tablet at night or a mild laxative water before breakfast. Flatulence and acid eructations may be controlled by sodium bicarbonate, but the fermentation giving rise to these symptoms is often best prevented by the use of hydrochloric acid.

ULCER OF THE STOMACH
(Peptic Ulcer. Round Ulcer)

In addition to such ulcers as those of tuberculosis, syphilis and typhoid fever, which are all extremely rare in the stomach, there exists an important and relatively common form—the peptic ulcer—which is peculiar to the stomach and duodenum and whose causation is still uncertain and obscure.

Etiology.—The disease is met with most frequently between the ages of twenty and thirty years, although it is not uncommon during the next decade and may occur at any age. It was formerly thought to be much more frequent in women than in men. Chlorotic girls of the servant class seem especially susceptible. It is sometimes met with in shoemakers, tailors and others whose occupation leads to continued pressure upon the epigastrium. It has been known to follow blows and other traumata of the abdomen. Of patients requiring surgical intervention the great majority are men.

PATHOGENESIS.—The origin of the gastric ulcer is still uncertain. It is generally believed that the direct cause is the digestion of a portion of the mucosa by the gastric juice, but the reason for such self-digestion is often not clear. In some, but by no means all, of the cases this is due to thrombosis or embolism of the arterial branch supplying the

affected region. Changes in the character of the blood, such as chlorosis and other forms of anemia, and hyperacidity of the gastric juice are regarded as important predisposing causes. Local infection by bacteria has been suggested as a possible cause.

Morbid Anatomy.—The ulcers are usually single, but two or more are not infrequently seen. They occur usually in the pyloric region and most frequently on the posterior wall near the lesser curvature. In the great majority of cases subjected to operation for ulcer the lesion is found not in the stomach but in the duodenum (80 per cent. of the cases operated upon by the Mayo brothers). Two forms, the acute and the chronic, are recognized. The acute ulcer is usually small (from one-third to one inch in diameter) and round, and has clean-cut, "punched-out" edges and a smooth base. The chronic ulcer is often larger, the outlines are irregular, the edges much thickened and indurated and the floor uneven and often terraced. The floor may be formed by the submucous, muscular or peritoneal coat or by the surface of some adherent organ such as the pancreas or liver. If the ulcer heals, a scar of varying extent and thickness is left which may result in obstruction of the pylorus or, rarely, in hour-glass constriction of the stomach. Instead of healing, however, it may perforate the stomach wall. Such perforation, especially if on the anterior surface, may result in general peritonitis, or it may cause a localized abscess, fistulous communication with the colon or duodenum or rarely perforation of the pleura or pericardium.

Symptoms.—Dyspeptic symptoms, vomiting, pain and hematemesis constitute the usual symptoms.

PAIN.—This is the most constant symptom and is rarely altogether absent, although it varies much in character and severity in the different cases. In its most characteristic form it is a severe, boring pain coming on a short time (from 15 minutes to an hour) after eating and felt in the epigastrium and back. It may persist for some hours, but is usually relieved by vomiting and sometimes by lying in a certain position, e.g.. upon the left side. It is usually, but not always, made worse by pressure upon the epigastrium.

VOMITING is seen in most cases. It may be severe and constant or may occur only infrequently. Usually it takes place an hour or more after eating. The vomited matter commonly contains an excessive amount of HCl and may contain blood.

HEMATEMESIS occurs at some time in about one-half of the cases. If, as is often the case, the bleeding is profuse, the blood will be vomited at once and in an unaltered condition. When the bleeding is slight and gradual it may be retained for some time and is then decomposed by the gastric juice and when vomited has the brownish black color and a grumous consistence which suggest coffee-grounds. The hemorrhage may be so severe as to cause death, or frequent smaller hemorrhages may result in severe anemia. Often the blood passes through the pylorus in quantities sufficient to give the characteristic "tarry" appearance

to the stools. In almost all cases traces of blood may be found in the stools by careful chemical tests.

The DIGESTIVE DISTURBANCES vary much and include epigastric weight and discomfort, acid eructations, nausea, constipation, etc.

PHYSICAL SIGNS.—Pressure usually reveals a circumscribed area of tenderness at some point in the epigastrium and often also a tender spot just to the left of the spine and opposite the tenth, eleventh or twelfth dorsal vertebra. Rarely the local thickening and induration about an ulcer of the anterior surface may be felt through the abdominal wall.

The CONSTITUTIONAL SYMPTOMS show great variation. In some cases the general nutrition and strength are well preserved. In others the anemia, wasting and weakness may be very severe. Fever is usually lacking. The GASTRIC CONTENTS or VOMITUS usually show an excess of HCl, but normal or diminished acidity does not exclude the presence of ulcer.

FIG. 16.—An oval gastric ulcer showing at its center an eroded artery from which hematemesis had resulted. The soft, cloudy appearance is normal to the gastric mucous membrane. From the collection of Dr. Walter B. James.

Course and Complications.—Some of the cases run an acute course which may end in recovery or may result in death from hemorrhage or perforation. (See Fig. 16.) Occasionally such hemorrhage or perforation may be the first evidence of the disease. In a majority of the cases, however, the course is very chronic, the symptoms persisting, often with periods of temporary improvement, for a number of years.

Perforation occurs in about 2 per cent. of the cases. (See Fig. 17.) If the ulcer be upon the anterior surface perforation usually results in acute general peritonitis. If upon the posterior surface a localized, subdiaphragmatic abscess usually follows. Perforation may occur into the pleura or pericardium. *Pyloric obstruction* with secondary dilatation of the stomach may follow the scarring of a healed pyloric ulcer. Adhesive perigastritis is a not uncommon complication.

Prognosis.—The mortality varies between 5 and 15 per cent., most of the deaths being due either to hemorrhage or to perforation.

Diagnosis.—Gastric ulcer may be confused with the gastralgia of nervous dyspepsia, with the gastric crises of tabes, with gall-stone colic, with gastric cancer and with hemorrhage from cirrhosis of the liver. The characteristic pain (excited by eating and relieved by vomiting), the vomiting, the hyperacidity, the hemorrhages and the prevalence of ulcer in chlorotic young women are the features of special diagnostic importance.
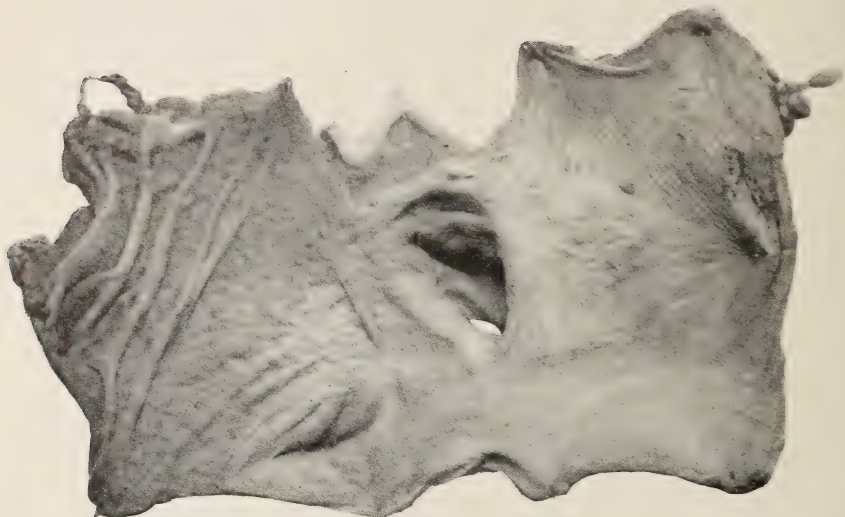
Fig. 17.—An old duodenal ulcer showing a perforation from which a subphrenic abscess had resulted. From the collection of Dr. Walter B. James.

Treatment.—The essential feature of this is to give to the stomach as nearly complete functional rest as possible. Absolute rest in bed for from three to four weeks should be insisted upon.

Diet.—If the symptoms are severe no food should be given by mouth, and rectal feeding should be resorted to for several days at least. (See Appendix, p. 587.) When mouth-feeding is resumed only the simplest food should be given, such as peptonized milk, milk and lime-water, albumin-water and meat broths, and these in small amounts frequently repeated. Gradually such articles as junket, meat jelly, custard and gruel may be added and later light and digestible solid food. Proteids, because of their tendency to cause free secretion of the acid gastric juice, should be used sparingly.

The Lenhartz diet makes practical application of these principles. The feedings are hourly for ten days, then every two hours, except from 9 p.m. to 7 a.m., during which time they are omitted. Water is given in moderate quantities.

LENHARTZ DIET

Day	Egg	Milk	Sugar per Day	Additional Feedings
1...	2 drams	4 drams	....	
2...	3 drams	6 drams	....	
3...	4 drams	1 ounce	....	
4...	5 drams	1½ ounces	5 drams	Sugar is added to the egg
5...	6 drams	1½ ounces	1 ounce	
6...	4 drams	2 ounces	10 drams	Scraped beef, 3 dr. to each of 3 feedings
7...	4 drams	2 ounces	10 drams	Boiled rice, 1 dram to each of 3 feedings
8...	4 drams	2½ ounces	10 drams	
9...	4 drams	2 ounces	10 drams	Rice, 2 dr.; dried toast, 5 dr.; 2 feedings
10...	4 drams	2 ounces	10 drams	Chicken, 1 ounce; butter, 5 drams
11...	....	6 ounces	....	

DRUGS.—Of the many drugs used bismuth subnitrate in large doses (ℨi several times a day) and silver nitrate (gr. $\frac{1}{4}$–$\frac{3}{4}$ t. i. d.) seem to be the most efficacious. Pain is often relieved by alkalies, but may require morphine. Vomiting is best treated by withholding from the stomach all food for a short time. For hemorrhage, absolute rest, morphine and rectal feeding are required. Drugs are of very doubtful value in checking hemorrhage. If the anemia is severe, iron, arsenic and other tonics are needed. Lavage is usually contra-indicated in gastric ulcer. Surgical treatment is indicated (1) for perforation; (2) for severe and repeated hemorrhages; (3) in certain chronic cases in which pain and other symptoms are severe and show no response to medical treatment, and (4) for cicatricial stenosis of the pylorus.

CANCER OF THE STOMACH

Etiology.—The stomach is, next to the uterus, the most common site of carcinoma. Gastric carcinoma, like other forms, is met with chiefly in persons of over forty years, but it is occasionally seen between the ages of thirty and forty and, rarely, also before thirty. Men are more frequently affected than women. A family history of cancer is obtained in a small proportion of the cases, but heredity is certainly not an important factor. Alcoholism and chronic dyspepsia also seem to have slight if any influence. Occasionally the disease develops in the wall of an old gastric ulcer. The cause of cancer is still entirely unknown.

Morbid Anatomy.—The growth may occur at any part of the stomach, but in almost two-thirds of the cases it is found in the pyloric region. It attacks the posterior surface much more frequently than the anterior, and the lesser curvature more often than the greater. In 8 per cent. of the cases the growth appears in the region of the cardia.

VARIETIES.—Four types of carcinoma are met with: the SCIRRHOUS, the MEDULLARY or encephaloid, the cylindrical-celled ADENO-CARCINOMA and the COLLOID form. The scirrhous type usually forms a firm, nodular mass near the pylorus which frequently causes stenosis; rarely it shows itself as a diffuse fibrous thickening of the stomach wall; the medullary

form and the adeno-carcinoma produce soft, rapidly growing vascular tumors which project into the lumen of the organ and tend early to break down and to ulcerate; colloid cancer causes a diffuse infiltration of all the coats and tends to early involvement of neighboring structures with growths of the same gelatinous nature.

Extension of the disease occurs (1) by the direct involvement of adjacent tissues and organs and (2) by metastasis. These metastatic deposits occur in a large proportion of the cases and are seen in the neighboring lymph-glands, in the liver, intestines, omentum, pancreas, lung, etc.

SECONDARY CHANGES.—There is usually a well marked chronic gastritis. If the pylorus be obstructed marked dilatation of the stomach frequently follows. If the growth obstruct the cardia the stomach is usually small and the esophagus dilated. Adhesions often occur between the stomach and the adjacent organs, and perforation occasionally results from extensive ulceration.

Symptoms.—These are both general and local:

GENERAL SYMPTOMS.—The *loss of flesh* is progressive and marked (amounting often to 50 or 60 lbs.) and is associated with great loss of strength, a waxy pallor and a more or less severe secondary anemia. There is often some edema of the legs in the late stages. Moderate fever is sometimes seen and there may even be chills. Death occurs usually from exhaustion, but it may result from a peculiar type of coma (*coma carcinomatosum*).

GASTRIC SYMPTOMS.—ANOREXIA is a very constant and persistent symptom in most cases and is often accompanied by *nausea*.

PAIN is present in most cases and is frequently the most troublesome symptom. It is usually a more or less continuous dull or gnawing pain which lacks the intermittence and the severe paroxysmal character of the pain of ulcer and which bears a less definite relation to the taking of food. It may be referred to the epigastrium or to the back or shoulder.

VOMITING occurs at some time in almost all cases. It may appear early and be persistent and intractable, or it may occur only in the latter part of the disease and then only occasionally. It is especially troublesome when there is obstruction of either the cardiac or pyloric orifice.

HEMORRHAGE.—Blood is found in the vomitus in about one-half of the cases. The bleeding is usually small in amount and for that reason the blood is retained in the stomach for some time and undergoes chemical change, so that when vomited it has a dark brown or black color and resembles coffee-grounds. Rarely large quantities of bright blood may be vomited. If blood reaches the intestine in any quantity it gives a black, "tarry" appearance to the stools. Recently it has been found by delicate chemical tests that minute quantities of blood can be demonstrated almost constantly in the feces in cancer of the stomach.

EXAMINATION OF THE STOMACH CONTENTS.—Digestion is usually slow and imperfect. If there is obstruction at the pylorus stagnation

and fermentation of the food may be marked. Fragments of tumor tissue, as well as blood, may occasionally be found in the vomitus or stomach washings. In the great majority of cases, after a test-meal, free HCl is absent and lactic acid is present. Free HCl is occasionally found, however, especially in those cases of carcinoma developing upon an old ulcer. A very long, non-motile bacillus—the Oppler-Boas bacillus —is regularly found when lactic acid is present.

It has been shown that cancer tissue contains a ferment capable of digesting proteids beyond the peptone stage. Upon this fact the glycyl-tryptophan test for cancer of the stomach has been proposed. In the presence of a peptid-splitting ferment this substance is split into its two constituents, glycyl and tryptophan, and the presence of the latter is demonstrated by the rose-color produced on the addition of bromide water.

The ordinary Ewald test-breakfast may be employed, or instead white bread-toast or biscuit with a glass of sweetened hot water. The test requires special training and experience. The presence of saliva, bile, or blood may vitiate the test, and its value is still in doubt.

PHYSICAL SIGNS.—Careful inspection and palpation of the abdomen will in many cases reveal the presence of a hard, nodular tumor which is usually located in the epigastrium, but may be situated in almost any part of the abdomen, and which is often very freely movable. It is usually more or less tender on pressure. Peristaltic waves are often to be seen and felt. Dilatation of the stomach will give its usual physical signs. The liver and subcutaneous lymph nodes should be examined for metastatic deposits.

Complications.—In the majority of cases metastatic growths occur in some of the neighboring organs and these, especially when in the liver, may give symptoms which altogether mask those of the primary disease. The possibility of profuse hemorrhage has been mentioned. Occasionally perforation and peritonitis result from ulceration of the growth.

Course and Termination.—The disease usually lasts from six months to two years. Acute cases sometimes run their course in three or four months. Although quite marked temporary improvement is sometimes seen the disease is always fatal.

Diagnosis.—Chronic gastritis, gastric ulcer, pernicious anemia and cirrhosis of the liver are the conditions most likely to be confused with gastric cancer. The advanced age, the progressive emaciation, the presence of a tumor, the vomiting of blood, the absence of free HCl, presence of lactic acid and the absence of characteristic blood changes are the features which point most strongly toward cancer. The diagnosis, however, cannot always be made immediately and it occasionally happens that the disease runs a latent or masked course throughout.

Treatment.—Complete removal of the growth by surgical means offers the only hope of cure and this is possible only in carefully selected cases and when the diagnosis is made very early. Aside from this the

treatment is only palliative and symptomatic. Systematic lavage will often relieve the dyspeptic symptoms, especially if pyloric obstruction exists. The pain may require the use of morphine. The operation of gastro-enterostomy, although only palliative, is sometimes of real service in the case of pyloric tumors in that it often relieves the pain and the tendency to hemorrhage and seems to delay somewhat the growth of the tumor.

ACUTE DILATATION OF THE STOMACH

Etiology.—While the possibility of acute dilatation of the stomach has been known for years, its clinical frequence and importance have only recently been recognized. A study of 102 cases by L. A. Conner, published in 1907, brought general attention to the subject in this country, and led to the more frequent recognition of this condition. It has been found in various relations: (1) After operations, particularly abdominal operations, under general anesthesia. (2) During convalescence from severe or wasting disease. (3) Following injuries of various kinds. (4) After distention of the stomach by large quantities of food or drink. (5) In disease of the spine, especially in patients wearing plaster jackets. (6) Without assignable causes in patients apparently in good health.

Morbid Anatomy.—In fatal cases the stomach and the first and second portions of the duodenum are greatly distended, the enlarged stomach filling the whole left side of the abdomen, and even reaching the pelvis. A constriction of the third portion of the duodenum as it crosses the vertebræ by the root of the mesentery containing the superior mesenteric artery is frequently found. The duodenal obstruction is brought about by downward traction upon the root of the mesentery. The gastric distention is attributed to the swallowing of air—aërophagia.

Symptoms.—The clinical picture is that of sudden intestinal obstruction, with repeated vomiting of bile-tinged fluid, abdominal distention and tenderness, constipation, thirst, scanty urine, and rapid collapse. Any one of these symptoms may be absent. The temperature is normal or below, the vomitus is bile-tinged, and may have a foul odor, but is not fecal. Distention of the epigastrium and left half of the abdomen with tympany, and often splashing sounds or fluctuation below the umbilicus, are important physical signs.

Prognosis.—Of the recorded cases about 75 per cent. proved fatal, but doubtless many mild cases recover spontaneously, and prompt recognition and proper treatment should insure recovery.

Treatment.—The stomach should be emptied by the stomach tube, and nothing given by mouth for a time. The patient should lie upon the abdomen, or upon the right side to relieve the duodenal stricture by relaxing the mesentery. Elevation of the foot of the bed may be helpful. Feeding by mouth and the ordinary position should be gradually resumed.

CHRONIC DILATATION OF THE STOMACH
(Gastrectasis)

Etiology.—Chronic dilatation of the stomach is of two types—obstructive and non-obstructive. The *obstructive form* results from a narrowing of the pylorus or duodenum which may be due to such intrinsic causes as cancer, cicatricial stricture of a healed ulcer, sclerotic thickening (hypertrophic stenosis), congenital stenosis and possibly pyloric spasm; or to such *extrinsic* causes as the pressure of a tumor, peritoneal adhesions, kinking of the pylorus, etc. The *non-obstructive form* results from muscular insufficiency due to habitual over-eating and drinking (as in diabetics, beer drinkers, etc.) or to chronic debilitating diseases, severe acute diseases (typhoid, etc.) and chronic inflammation of the stomach wall.

Morbid Anatomy.—In the obstructive type there is at first an hypertrophy of the muscular coat which may for a time compensate for the narrowing of the orifice. Eventually, however, the stomach becomes much increased in size and the muscular coat thin and atrophic. The mucosa usually shows a well-marked chronic gastritis.

Symptoms.—These depend upon the imperfect emptying of the stomach; the decomposition of the retained food, and the associated chronic gastritis. The symptoms develop gradually and consist of a feeling of weight and dragging or of dull pain in the stomach, flatulence, eructations of gas, and especially the HABITUAL VOMITING at longer or shorter intervals OF VERY LARGE QUANTITIES OF FLUID AND FOOD. In the vomited matter there are often to be seen remnants of food eaten many hours or even a day or two before. The delay in the emptying of the stomach may be demonstrated also by passing a stomach tube before breakfast, when food eaten the evening before can be recovered. The vomitus has an offensive, sour odor and contains usually a variety of fungi and bacteria. The appetite is usually lost, but there may be constant hunger. The bowels are obstinately constipated, nutrition is usually much impaired and, because of the diminished absorption of fluid, there are usually thirst, scanty urine and dryness of the skin. Vertigo and headache are common. Tetany is an occasional complication.

Chemical analysis of the stomach contents after a test-meal, and of the vomited matter, shows usually great diminution or absence of free HCl and the presence of organic acids (lactic, butyric and acetic) due to the abnormal fermentative processes. Occasionally HCl may be normal or increased in amount.

The ability of the stomach to empty itself, impaired as a rule either by lessened motility of its wall or by obstruction of the pylorus, is most simply tested by giving with the evening meal half a dozen raisins or a tablespoonful of cranberry or currant preserve. If there is retention, the seeds and skins of the fruit will be obtained on washing out the stomach in the morning.

PHYSICAL SIGNS.—In the dorsal position INSPECTION will often show

a fullness in the abdomen corresponding to the enlarged stomach, and the outline of the greater curvature may be seen lying *below* the umbilicus (normally it lies a finger's breadth or more above).   If there be pyloric obstruction waves of peristalsis may frequently be seen passing from left to right across the abdomen.   By PALPATION a splashing sound is easily elicited even though no food or drink has been taken for several hours, and the splash can be produced at points beyond the normal limits of the stomach.   Pyloric tumors or thickening may be felt, and the peculiar elastic feel of the dilated stomach itself may often be recognized through the thin abdominal wall.   By PERCUSSION the outlines of the enlarged stomach may be determined accurately if the organ be inflated with air or gas.   This may be done by means of a stomach tube and a Davidson's syringe, or by the use of tartaric acid and sodium bicarbonate (āā 3ss–3j) each dissolved in 3ij of water and taken separately.   In the erect position the most dependent part of the stomach will yield a dull note, from the contained fluid, and this will be found to be on a level with or below the umbilicus.

Diagnosis.—For this it is necessary to prove, first, that the stomach is enlarged and, second, that food is retained in it beyond the normal time (seven hours or less).   The condition is to be distinguished from an unusually large stomach (*megalogastria*) in which the motor function is not impaired, and which therefore gives no symptoms; from *atony* of the stomach (as shown by a delay in emptying itself) *without enlargement,* and from *gastroptosis* in which the stomach is displaced and lies abnormally low in the abdomen.

RADIOGRAPHIC EXAMINATION of the stomach filled with a bismuth emulsion will show its size and position.   Retention of the bismuth may also be demonstrated by examination 24 hours later.

Prognosis.—This depends not only upon the nature of the cause, but also upon the degree of dilatation and the condition of the muscular coat. Non-obstructive cases of moderate severity are often greatly benefited or altogether cured by careful treatment.   The prognosis of cases with non-malignant stenosis of the pylorus has been much improved by the recent rapid development of gastric surgery.

Treatment.—The cause should be carefully sought for and, in so far as possible, removed.   The diet should be simple and nutritious and should be supplied in the form of five or six small meals.   Large meals and large quantities of fluid are distinctly injurious.

LAVAGE is often of very great value.   It should be performed daily, preferably at bedtime, and may need to be continued for many weeks or months.   The fluid used may be warm water or some mild alkaline or antiseptic solution.

Hydrochloric acid is often of service in aiding digestion and in diminishing fermentation.   Strychnine and other tonics and abdominal massage are of value in improving the muscular tone.

If marked pyloric obstruction exist little beside palliation can be expected from medical treatment and recourse should be had to surgery,

which of late has effected many brilliant cures. The relative merits of pyloroplasty and gastro-enterostomy cannot here be discussed.

Gastroptosis.—See Enteroptosis.

CONGENITAL HYPERTROPHIC STENOSIS OF THE PYLORUS

Definition.—Narrowing of the pylorus due to congenital hypertrophy of its muscular coats.

Etiology.—The cause is not known. Sometimes other congenital anomalies accompany the pyloric stenosis. Hypertrophy from overwork, due either to lack of proper nervous co-ordination or to irritation produced by improper food or hyperacidity, has been suggested, but not proven.

FIG. 18.—Hypertrophic pyloric stenosis. Section through the pylorus and adjacent duodenum. *M*, mucous membrane; *S M*, submucous coat; *C M*, circular muscular coat; *L M*, longitudinal muscular coat. Dr. John Dorning's case.

Morbid Anatomy.—The pylorus is found thickened and enlarged, often forming a definite tumor, 2 or 3 cm. in length and 1 in width. (See Fig. 18.) The lumen is greatly restricted. On section the wall is notably thickened by hypertrophy of the muscularis. The other coats are normal. The stomach is more or less dilated and consequently thinned.

Symptoms.—These appear within three months of birth. (a) Vomiting at first occasionally, later after every feeding, no matter what its composition; (b) obstinate constipation, with scanty, hard, dry stools; (c) and emaciation are the striking symptoms. Constant crying from hunger is naturally common.

On examination one finds (a) more or less extreme emaciation; (b) an abdomen distended, especially in the epigastrium; (c) peristaltic waves passing from left to right in the wall of the dilated stomach; (d) possibly a tumor just to the right and above the umbilicus. The

tumor is small, smooth, painless, freely movable; (e) washing out the stomach relieves the distention.

Diagnosis.—The combination of symptoms and physical signs is diagnostic. If no tumor be found, it is possible that the symptoms are due to pyloric spasm without hypertrophy and may yield to careful feeding and repeated lavage.

Prognosis is always grave. Mild cases may recover without operation, but most require it.

Treatment.—Careful feeding with a low-fat milk mixture suited to the age of the child may be tried. Absolute quiet after feeding is essential. Breast-feeding is most favorable, but the time of nursing must be limited.

Lavage, once or twice daily, should be employed.

Operative relief is usually required and should not be unduly delayed. Gastro-enterostomy, pyloroplasty, and divulsion (Loreta's operation) have been tried. The first operation is usually preferred.

HEMORRHAGE FROM THE STOMACH
(Gastrorrhagia, Hematemesis)

Etiology.—Vomiting of blood (*hematemesis*) may result from bleeding into the stomach itself (*gastrorrhagia*) or from the accumulation there of blood from some other source, *e.g.*, nose, pharynx, esophagus or duodenum.

Gastrorrhagia may result from (1) *Local causes* such as ulcer, cancer, traumatism, severe gastritis and miliary aneurisms of the small arteries. (2) *Passive congestion* of the stomach due (a) to cirrhosis of the liver; (b) to thrombosis of, or pressure upon, the portal vein; (c) to great enlargement of the spleen, and (d) to venous stasis in chronic disease of the heart. (3) *Changes in the character of the blood*, as seen in such toxic states as yellow fever, small-pox, measles, cholemia and phosphorus poisoning, and in such constitutional diseases as the severe anemias, leukemia, purpura, scurvy, hemophilia, etc.

In cirrhosis of the liver a common source of the vomited blood is the varicose veins which are so prone to form at the lower end of the esophagus.

Symptoms.—In general the larger the hemorrhage the more promptly is it vomited. Rarely even a large hemorrhage may be retained and passed into the intestine. The vomited blood is usually dark colored and more or less clotted. If it has remained in the stomach for some time it is apt to have the brownish black, grumous appearance of coffee-grounds. Single hemorrhages are sometimes seen, but more frequently the bleeding continues interruptedly for several days or more. The symptoms of anemia vary with the severity of the hemorrhage.

Diagnosis.—Vomited blood is usually readily recognized, but small amounts of changed blood may require the aid of the microscope or of the chemical or spectroscopic tests. The nose, mouth and throat should

be carefully examined for evidence of recent hemorrhage in view of the possibility that the blood may have been swallowed. Occasionally it may be difficult to decide at first whether the blood has come from the stomach or the lungs. In the former case the blood is usually dark and clotted, and may be mixed with the acid stomach contents; the vomiting usually occurs without cough, the stools may later contain blood and there are other evidences of gastric, hepatic or splenic disease. In the case of hemoptysis the blood is brought up by coughing, is bright, frothy and fluid and is alkaline in reaction. The sputum is subsequently blood stained; moist râles may be heard over some part of the chest and there are other evidences of disease of the lungs or heart.

Treatment.—Absolute rest; the withholding of all food and drink by mouth and morphine subcutaneously are the essential features. Water may be given by rectum or by hypodermoclysis. The value of local astringents and of ice to the epigastrium is questionable. Stimulant drugs, hypodermoclysis or saline infusions should not be used unless life is actually threatened by exsanguination. After twenty-four or forty-eight hours feeding may be gradually resumed, as in gastric ulcer.

NEUROSES OF THE STOMACH
(Nervous Dyspepsia)

It has been conclusively proved that most of the symptoms of gastric disturbance may be produced by purely functional causes and without the existence of any recognizable pathological changes in the stomach. Such functional disturbances are grouped under the general name of *nervous dyspepsia* and may closely simulate those caused by such organic lesions as chronic gastritis, ulcer and cancer. They are seen most commonly in young adults, especially women, and in those of neurotic and emotional temperament, and are usually only a part of a general neurasthenia which, however, may be almost altogether masked by the prominence of the gastric symptoms.

The gastric neuroses may be sensory, secretory or motor in character. These types sometimes occur singly and distinct, but frequently they are combined in various ways to make up the complex picture of nervous dyspepsia.

Sensory Neuroses.—(a) GASTRALGIA; GASTRODYNIA.—This neurosis presents a very distinct clinical picture characterized by definite paroxysms of intense epigastric pain, lasting from a few minutes to several hours, and not accompanied, usually, by vomiting or other gastric disturbance. The pain may radiate to the back or shoulders and is often somewhat relieved by pressure. Following the attack there is frequently the passage of a large quantity of pale urine. The attacks usually bear little or no relation to the taking of food, but are apt rather to be precipitated by such psychical causes as excitement, anger, etc. The HCl of the stomach may be normal or increased in amount. The intervals between the attacks vary greatly in length, but often last for months

and during this time there are usually no symptoms. Gastric ulcer, **gall-stone** colic, angina pectoris and the gastric pains of tabes dorsalis are the conditions most apt to be confused with gastralgia.

(b) HYPERESTHESIA OF THE MUCOUS MEMBRANE.—Abnormal sensitiveness of the stomach mucosa to food is the feature of this neurosis. This may be shown to all food or only to special dishes. Similar pain, burning and distress after eating is seen, of course, in various organic diseases and these must be carefully excluded before the diagnosis can be made.

(c) DISTURBANCES OF THE APPETITE.—There may be complete loss of appetite (*anorexia nervosa*), as seen at times in hysteria, melancholia, chlorosis, etc.; or abnormally great hunger (*hyperorexia, bulimia*); or rarely, the loss of the sense of repletion after eating (*akoria*).

Secretory Neuroses.—(a) NERVOUS HYPERCHLORHYDRIA (HYPER-ACIDITY).—The secretion of gastric juice containing an excessive amount of HCl, which is so constant a feature in gastric ulcer, is seen also as a pure neurosis. The symptoms appear usually from one to four hours after eating and consist of epigastric pain and burning, acid eructations, thirst and often excessive hunger. The pain is usually promptly relieved by taking of proteid food (*e.g.,* milk) or an alkali, or by vomiting. Gastric digestion is prompt and complete. Constipation is common.

(b) GASTROSUCCORRHEA (SUPERSECRETION).—In this condition there is a more or less constant and abundant secretion of acid gastric juice *even when the stomach is quite empty of food.* It is seen in both a *periodic* and a *continuous* form. Epigastric distress, burning and pain, acid eructations, headache and the vomiting of large quantities of clear, yellow, acid fluid are the features. By the use of the stomach tube considerable quantities of this fluid can be obtained from the fasting stomach.

(c) NERVOUS SUBACIDITY OR ANACIDITY.—Diminution of the acid of the gastric juice is a common manifestation of nervous dyspepsia. Rarely there may be complete absence of all gastric secretion including the ferments (*achylia gastrica nervosa*). It is characteristic of these functional derangements of gastric secretion that the findings of stomach analysis are inconstant and varying. At one time there may be diminishing acidity, at another complete absence of free HCl and at another normal gastric juice.

Motor Neuroses.—(a) HYPERKINESIS, PERISTALTIC UNREST.—Excessive muscular activity of the stomach is frequently associated with secretory neuroses (hyperchlorhydria, achylia) and occurs rarely as a primary neurosis (*peristaltic unrest*), in which case the active peristaltic movements are felt not only during digestion, but also when the stomach is empty.

(b) NERVOUS ERUCTATIONS AND VOMITING.—Paroxysms of noisy eructation of gas constitute a rather common neurosis of hysterical and neurasthenic persons. The gas consists chiefly of air which is unconsciously swallowed.

Vomiting not due to any organic change in the stomach is seen in various affections of the central nervous system (brain tumor, tabes, etc.), and occurs also as a manifestation of hysteria.

(c) FUNCTIONAL DISTURBANCES OF THE CARDIA AND PYLORUS.—Painful *spasm* of the cardia is seen rarely as a primary neurosis. There may be *insufficiency* of the cardia, with the habitual regurgitation of gas or food. If the regurgitated food is again masticated and swallowed the condition is known as *rumination*.

Pyloric spasm, which is so often seen in gastric ulcer, occurs also in the purely functional hyperchlorhydria, and in hyperesthesia of the mucous membrane. Functional insufficiency of the pylorus, also, is recognized.

Treatment of Neuroses of the Stomach.—Treatment of the underlying nervous state is of primary importance. Rest, out-of-door exercise, travel, etc., may be all that is needed in some cases. In others a carefully regulated "rest cure" may be advisable. Tobacco, alcohol, tea and coffee should be used very sparingly if at all. The troublesome pain of hyperchlorhydria is best relieved by proteid food and by large doses of alkalies—bismuth, magnesia, or sodium bicarbonate. In gastrosuccorrhea daily lavage, atropine, nitrate of silver (gr. $\frac{1}{4}$–$\frac{1}{2}$), and the alkalies are of value. In the motor neuroses small doses of sodium bromide and chloral are often useful. If anemia be present arsenic and iron should be used.

DISEASES OF THE INTESTINES

I. ENTERITIS

(Catarrhal Enteritis. Catarrhal Entero-Colitis)

Inflammation of the intestinal mucosa occurs in both an acute and a chronic form, the former being much the more common.

Etiology.—Acute catarrhal enteritis is of very frequent occurrence and may be either *primary* or *secondary*. The causes of the primary form are: (a) Improper food, such as unripe or over-ripe fruit, tainted meat or milk, etc.; (b) violent purgatives and certain mineral and other poisons; (c) impure drinking water, and (d) sudden chilling of the body and exposure to cold. The *secondary* form occurs in (a) certain infectious diseases, such as typhoid, pneumonia, cholera, etc.; (b) passive congestion from cardiac disease or cirrhosis of the liver; (c) various cachectic conditions.

Morbid Anatomy.—The inflammation may affect only a portion of the small or the large bowel alone, but as a rule a considerable portion of the whole intestinal tract is involved (entero-colitis). The changes are those of catarrhal inflammation of any mucous membrane; hyperemia, exudation of serum and leukocytes (swelling) and an increased secretion of mucus. The hyperemia is rarely apparent after death. Swelling of the solitary and agminated follicles is very common, especially in children, and the summits of these swollen follicles may

become eroded (follicular ulcers). In chronic cases the mucosa may be thickened by the formation of new connective tissue or may be thinned and atrophic.

Symptoms.—The disease may be *acute* or *chronic;* the former being much the more frequent. *Diarrhea* is usually the chief and sometimes the only symptom. It is occasionally lacking, however, when the inflammation is confined to the upper part of the small gut. The stools vary greatly in appearance and in number. Their consistence is usually watery, the color varies from dark brown to a light yellow or gray, and the number from two or three to fifteen or twenty a day. Frequently particles of undigested food or shreds of grayish or bile-stained mucus are to be seen. Colicky pains and rumbling noises (borborygmi) are common and there may be slight general abdominal tenderness. Tenesmus and straining occur when the lower part of the large bowel is involved. Fever is slight or lacking. Anorexia, thirst and a coated tongue usually accompany the intestinal symptoms. In severe cases the prostration is marked and may amount to actual collapse. The duration of the acute cases is usually from two to five days.

Chronic catarrh may follow an acute attack, may result from passive venous congestion or certain stomach derangements, or may develop without known cause. Sharp pain is less common than in the acute form, but the nutrition often suffers severely and there may be great loss of flesh and strength. Many of the cases are marked by great mental depression. Diarrhea occurs often only at certain times of the day, *e.g.*, in the early morning or soon after eating.

Prognosis.—This in the acute cases is uniformly good. Only rarely does the inflammation persist and run a chronic course. In the chronic form the prognosis is by no means so favorable. Complete recovery is not common, although great improvement is often seen from suitable treatment. Frequently the symptoms persist for many years with alternating periods of improvement and exacerbation.

Treatment.—In the acute cases rest in bed, and a fluid or semi-fluid diet should be ordered. If the attack be due to dietetic errors a purge of castor oil or calomel should be given at the outset. The pain may be relieved by hot applications to the abdomen, spirits of chloroform ($\frac{7}{3}$j), small doses of opium; or, if severe, by morphine (gr. $\frac{1}{6}$–$\frac{1}{4}$) subcutaneously. For the diarrhea bismuth subnitrate (gr. xx–xxx), given after each loose stool, is usually all that is needed. Treatment of the *chronic* cases is by no means so simple. The cause should be carefully sought for and will often be found to lie in some derangement of the digestive processes in the stomach. Much information may often be obtained from careful examinations of the stools for undigested food, mucus, pus, blood, etc. In some cases rest in bed for a time and a carefully restricted diet may be all that is required. A strict milk diet is beneficial in some, but by no means in all cases. If the trouble seems to lie chiefly in the colon daily large irrigations of normal salt solution are often of value. The drugs most frequently found to be of benefit

are the bismuth salts (subnitrate, subgallate and salicylate), given in large doses; naphthaline (gr. x–xv), and capsules of salol (gr. v), and castor oil (℥v–x). The opium preparations and the strong astringents are rarely of permanent benefit. The constant use of a flannel abdominal band seems often to be helpful. Some cases are cured by change to a warm, dry climate.

Croupous or Pseudo-membranous Enteritis.—Croupous or diphtheritic inflammation of the intestines is seen (a) secondary to certain acute infectious diseases, such as pneumonia, typhoid fever, scarlet fever and pyemia; (b) as a terminal condition in such chronic affections as chronic nephritis, cancer, tuberculosis, cirrhosis of the liver, etc., and (c) in poisoning by minerals such as mercury, lead and arsenic. The process shows every grade of severity from a thin membranous film capping the tops of the folds of the mucosa to an intense gangrenous inflammation with extensive sloughing. The colon is usually the part chiefly affected. The symptoms may be those of a severe entero-colitis with mucus, pus, blood or shreds of false membrane in the stools, but frequently the local diarrheal symptoms may be masked by those of a severe general infection.

Phlegmonous Enteritis.—This extremely rare condition usually occurs as a complication of intussusception, strangulated hernia or intestinal ulceration. The purulent infiltration involves chiefly the submucous layer and may be circumscribed or more or less diffuse. The symptoms resemble those of acute peritonitis and usually end fatally.

CHOLERA NOSTRAS

(Cholera Morbus)

Etiology.—This disease is not very infrequent in temperate climates during the hot months. It occurs both in adults and in children and usually seems to be due to some bacterial toxin formed in tainted meat, shell fish, milk, ice cream, etc. In some cases no such cause is discoverable. The disease is not transmissible.

Symptoms.—The symptoms cannot be distinguished from those of Asiatic cholera. There is a sudden onset, with persistent vomiting and purging; colicky pains in the abdomen; cramps in the muscles of the legs; great thirst and often alarming prostration. The stools are very numerous and soon lose their fecal character, becoming serous and odorless. The attack lasts from a few hours to several days. Complete recovery is the rule. Only rarely does a case prove fatal. The post-mortem findings are in no way characteristic. There is usually only a moderate grade of catarrhal inflammation of the stomach and intestines with some swelling of the solitary and agminated lymph follicles.

Diagnosis.—Confusion with true cholera is not likely unless an epidemic of the latter disease prevails. In such cases the differentiation can be made only by careful bacteriological examinations of the stools.

The TREATMENT is similar to that described under Asiatic cholera.

II DIARRHEAL DISEASES OF CHILDHOOD

Etiology.—AGE.—Diarrhea is especially prone to attack children between the time of weaning and the end of the second year. In bottle-fed babies the condition is very common during the first year.

SEASON.—Most of the cases occur in hot weather and especially during the months of July and August. The heat seems to exert its baneful influence chiefly by favoring bacterial growth and thus hastening fermentative changes in milk and other foods, rather than by its direct effect upon the child itself. The degree of humidity has little or no influence.

DIET.—Diarrheal diseases are vastly more common in artificially-fed babies than in those nursed. This is particularly the case among the lower classes, where a shocking degree of ignorance and carelessness exists as to the selection and preparation of such artificial food. Impure or stale milk, unclean bottles and nipples, indigestible articles of food, over-feeding and the prolonged use of condensed milk and the various proprietary foods are common causes of disease.

STATE OF HEALTH.—Chronic indigestion, rickets, marasmus, syphilis, tuberculosis and all conditions which lower the child's vitality are important predisposing causes of intestinal disorders.

BACTERIOLOGY.—Although there can be little doubt that many cases of diarrhea in children, especially those occurring during the hot months, are instances of bacterial infection, and although the diarrheal stools contain many forms of bacteria not found in those of healthy infants, the causal relation of a particular organism to any special form of diarrhea has not yet been proven. Bacilli of the *colon* and *proteus* groups; the pyogenic cocci, and certain saprophytic bacteria such as the hay-bacillus, are the varieties usually found. Recently it has been shown that the *Bacillus dysenteriæ* of Shiga, or an organism very closely allied to it, is present in many cases of summer diarrhea of both mild and severe type, as well as in certain local epidemics of dysentery in children. Its significance is not yet fully determined.

CLINICAL TYPES *

1. **Acute Intestinal Indigestion.**—This condition regularly results from some dietetic error, such as the use of contaminated milk or of other improper food.

The SYMPTOMS depend upon the irritation of the food itself or the toxic materials contained in it and not upon any lesion of the intestinal mucosa. They usually begin quite suddenly and consist of colicky pains, flatulence, diarrhea and more or less constitutional disturbance. There may be vomiting. The stools number from four to ten in the 24 hours, contain curds and other undigested particles, have an acid reaction and

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* The classification of Holt is here followed.

a sour odor and, in infants, are of a green or yellowish-green color. Blood is never present and mucus is scanty or lacking. Moderate fever, restlessness, and prostration are present at the outset, but usually subside within a day or two under proper treatment. Most cases recover within three or four days, but in delicate and susceptible children, or in those not suitably treated, the condition may be the starting point of some more serious intestinal disorder.

2. **Acute Gastro-Enteric Infection (Fermentative Diarrhea).**—In this form, which prevails in hot weather, the symptoms are manifestly due to the absorption of toxic products resulting from abnormal fermentative processes in the stomach or intestine and not to local inflammatory changes, for these are usually slight and insignificant. To this form belong a large proportion of the cases of summer diarrhea. Two distinct classes of cases are seen:

(a) Simple Gastro-Enteric Infection.—The symptoms as a rule begin quite abruptly with vomiting, diarrhea, colicky pain and a rapid rise of temperature to 102°–105° F. In many cases nervous symptoms are prominent. There may be great restlessness and irritability or one or more convulsions; or, on the other hand, apathy and stupor. The pulse is rapid and weak and the prostration sometimes very great. The stools number from six to twenty a day. They are at first fecal, but soon become thin and fluid and show many undigested lumps of curd and fat. A special feature is the very offensive odor of the stools and the large amount of flatus which accompanies them. Blood is lacking and mucus is present in only small amounts, unless the trouble continue for a number of days. Most cases recover within a week or ten days. Some few die in collapse and some develop the lesions of ileo-colitis.

(b) Cholera Infantum.—This term is reserved for an intense and virulent form of infection or intoxication which fortunately is far from common. It usually attacks debilitated infants and those with existing digestive disorders. No characteristic lesions are found in the intestines. The clinical picture closely resembles that of true cholera. The onset is abrupt with persistent vomiting and purging, the stools being very frequent and soon consisting of little but pure serum. They have an alkaline reaction and are almost odorless. There is great restlessness or stupor, intense thirst, a high internal temperature with cold surface and extremities, a grayish pallor, pinched features, sunken eyes and fontanelles, a small and very rapid pulse and profound collapse. Loss of weight is extraordinarily rapid and marked. In some cases convulsions and symptoms suggestive of meningitis ("spurious hydrocephalus") occur. Many of the cases die of collapse within the first 24 or 36 hours; in some others the purging and vomiting cease, but the child passes into a state of stupor or coma and dies in the course of three or four days; some few recover completely and still others develop the symptoms of ileo-colitis.

3. **Ileo-Colitis (Entero-Colitis, Inflammatory Diarrhea).**—In this type of diarrhea the symptoms are associated with, and depend upon,

pronounced inflammatory changes in the intestinal mucous membrane. Such inflammation may supervene upon any of the forms of diarrhea already described or may be primary. The inflammation may be catarrhal, pseudo-membranous or ulcerative. The ulcers may be small and limited to the areas about the solitary lymph follicles (follicular ulcers) or may be very extensive and deep. The lower portion of the ileum and the colon are the parts usually chiefly involved. The severity of the symptoms corresponds to that of the intestinal lesions. There is a continued fever, loss of flesh and strength, abdominal pain and tenderness and diarrheal stools containing much mucus and often blood and pus. In the pseudo-membranous form the stools may contain shreds of false membrane. Rectal tenesmus and straining are common and may lead to prolapse of the gut. The duration of the disease varies from ten days to several weeks. The severest cases are marked by grave constitutional symptoms, high fever and death within one or two weeks. Many of the milder cases recover completely, but the disease is often very protracted and is marked by frequent exacerbations in the symptoms. Bronchopneumonia is a common and serious complication. In certain of the cases the disease finally assumes a *chronic form* in which there are progressive emaciation, a normal or subnormal temperature, diarrhea with mucous stools, tympanitis and a dry, red tongue. Abdominal pain and tenderness are usually absent. Such cases may last for many months, but most of them die eventually of exhaustion and malnutrition.

Prophylaxis.—The importance of careful attention to prophylaxis can hardly be overestimated since most of the diarrheal troubles in children are preventable. An abundance of fresh air and sunlight, cleanliness of body, frequent bathing and light clothing in hot weather and plenty of pure drinking water are essential to the child's health. Breast feeding should be insisted upon whenever possible and weaning avoided during hot weather. The importance of the proper selection of artificial food, of regularity in feeding, of not overfeeding and of scrupulous cleanliness of bottles, nipples, etc., should be impressed upon mothers and nurses. Throughout the hot months milk should be sterilized or pasteurized and the drinking water boiled, and in very hot weather the quantity and strength of the food should be reduced and an increased amount of water given. Every slight digestive ailment demands careful attention and the popular notion that in teething infants looseness of the bowels is normal and unobjectionable should be vigorously combated.

Treatment.—In almost every case of diarrhea when seen early there are two clear indications—first, to empty the intestines of decomposing and irritating materials and, second, to secure complete rest for the digestive organs. The first is best met by the use of calomel or castor oil. If there is much nausea or vomiting calomel is to be preferred. It may be given in gr. $\frac{1}{6}$ doses every hour for six or eight doses. The second indication is met by withholding all food for 12, 18 or even 24 hours and relieving thirst by small quantities of water given frequently. When feeding is begun only the simplest foods, such as whey, broths

albumin-water, malted foods, etc., should be used in small and frequent doses. After two or three days milk may usually be begun, but only in very dilute form. In most of the mild cases of intestinal indigestion or infection little else is required in the way of treatment. In the more severe cases the various symptoms may require special treatment. For the diarrhea the bismuth preparations—preferably the subnitrate (gr. x–xx q. 3–4 hrs.)—are to be recommended. The stronger astringents and the so-called intestinal antiseptics are usually much less satisfactory. If the diarrhea persist and especially if there be much colicky pain opium may be needed, but it should always be used with caution and should be avoided if possible. It may be given in the form of Dover's powder (gr. $\frac{1}{4}$–$\frac{1}{2}$), paregoric ($\mathfrak{m}$v–xv) or the deodorized tincture ($\mathfrak{m}\frac{1}{4}$–$\frac{1}{2}$) every two to four hours. The temperature and the nervous symptoms are best controlled by tepid baths or wet packs. If stimulation is required brandy ($\mathfrak{m}$x–$\mathfrak{z}$ss) and strychnine (gr. 1/500–1/100) in frequently repeated doses may be used.

In cholera infantum morphine subcutaneously (gr. 1/100–1/60) should follow promptly after an initial purge of calomel and should be repeated once or more if needed to control the purging and to neutralize the effect of the toxin upon the heart and nerve-centers. The enormous loss of fluid is best met by subcutaneous injections of several ounces of normal salt solution. Stimulants should be given hypodermatically and the fever controlled if possible by baths and cold water irrigations of the colon. Immediate removal of the child to the country or seashore will sometimes save an apparently hopeless case.

In ILEO-COLITIS, in addition to the measures described for gastro-intestinal infection, much benefit may be obtained from the daily irrigation of the colon with a large quantity of normal salt solution or some mild astringent. Drugs are of little value in these protracted cases and the most essential part of the treatment, aside from colon irrigations, lies in the careful regulation of the diet. Whenever possible the child should be sent to the mountains or seashore.

INTESTINAL ULCERS

Ulceration of the intestine occurs in a great variety of forms. The ulcers of typhoid fever, dysentery, tuberculosis, syphilis and cancer are described elsewhere.

Duodenal Ulcer.—This important form of ulcer seems to be identical in its mode of origin and its morbid anatomy with the round ulcer of the stomach. It occurs almost always in the first portion of the duodenum and within an inch or two of the pylorus. It has occasionally developed as a complication of severe burns and of chronic nephritis. It is seen much more commonly in men than in women and its period of greatest frequency is from the thirtieth to the fiftieth year.

The symptoms are very similar to those of gastric ulcer, and in many

cases it is quite impossible to differentiate between the two conditions. In general, however, pain is a less constant symptom in duodenal ulcer and when present is apt to occur later after meals or when the stomach is empty; vomiting is much less common; tenderness also is less frequent and is usually located in the right hypochondrium; hematemesis is not so often met with, whereas bloody stools (melena) are seen more frequently. A good many cases run a latent course and the first indication of any trouble may be a profuse intestinal hemorrhage or the signs of perforative peritonitis.

The treatment is that of gastric ulcer, and surgical intervention is often required.

Follicular ulcers are common in the ileo-colitis of children and are occasionally seen as a complication to various severe diseases in adults. They result from inflammation and erosion of the solitary follicles and occur chiefly in the large intestine.

Stercoral ulcers occasionally result from the irritation of scybalous masses in chronic constipation. Such ulcers not infrequently occur in the vermiform appendix and seem often to be the starting point of an acute appendicitis.

Solitary ulcers of unknown origin are met with rarely in both the large and small intestine and may result in perforation.

Ulcerative Colitis.—Ulcerative inflammation of the colon, quite distinct both etiologically and clinically from true dysentery, is not very uncommon. It is most often seen in people of middle age. The symptoms are those of diarrhea, pain, slight fever and loss of flesh. The stools may contain blood, but mucus is usually lacking, as are also tenesmus and rectal discomfort. The affection often runs a chronic course. Treatment is similar to that of dysentery.

CHOLERA

(Asiatic Cholera)

Definition.—An acute infectious disease occurring chiefly in great epidemics, caused by a specific micro-organism—the comma bacillus, and marked by serous vomiting and purging, painful cramps and collapse, and by a very high mortality.

Etiology.—Cholera is endemic in certain parts of India. From these centers it spreads from time to time in devastating epidemics over the whole of Asia, to many portions of Europe and occasionally to other parts of the world. It has several times visited this country. Infection occurs almost wholly through the medium of water (streams, wells, etc.) which has become contaminated by the discharges of cholera patients. Any derangement of the digestive tract seems to predispose to such infection. The specific germ is the comma bacillus of Koch, which is found in vast numbers in the intestines and stools, but does not occur in the blood or viscera. It is a small, comma-shaped, actively motile

organism which enters the gastro-intestinal tract with the drinking water or food and there multiples rapidly, producing at the same time a potent toxin to which the violent symptoms seem due.

Morbid Anatomy.—The body after death has a shrunken, livid look; often there is a high post-mortem temperature. Rigor mortis occurs very early and is marked, and post-mortem contraction of the muscles is frequently seen. The tissues are abnormally dry; the blood is thick, dark and liquid; the intestines contain large quantities of turbid serous fluid. The intestinal mucosa is sometimes blanched, sometimes congested. The solitary follicles and Peyer's patches are swollen and prominent. The heart, liver and kidneys, especially the last, are apt to show extensive parenchymatous degeneration.

Symptoms.—The INCUBATION PERIOD is rarely longer than three or four days and sometimes lasts only a few hours.

It is convenient to divide the symptoms into several stages which are more or less distinctly defined.

1.—THE STAGE OF PRELIMINARY DIARRHEA.—In many cases a mild diarrhea is present for a day or two before characteristic symptoms appear. In other cases the onset is abrupt with

2.—THE STAGE OF SEROUS PURGING.—There is violent purging. The movements soon lose their fecal character and consist of frequent, painless evacuations of very large quantities of watery fluid containing flakes of desquamated epithelium and fibrin—the so-called "rice-water stools." These are odorless and alkaline, are composed of almost pure serum and commonly contain neither blood nor mucus. With the purging there is usually also persistent vomiting of similar serous fluid. There are intense thirst and great restlessness. A striking feature of this stage is the very painful cramps, which affect chiefly the muscles of the lower extremities and abdomen. As a result of the enormous loss of body fluid there follows, usually within twenty-four hours,

3.—THE ALGID STAGE, OR STAGE OF COLLAPSE.—In this the purging and vomiting may cease. The skin is cyanotic and clammy, the cheeks and eyes sunken, the voice whispering, the urine, bile and other secretions are suppressed and the pulse feeble and flickering. The surface temperature is much below normal, but the internal temperature is usually somewhat elevated.

If this period is survived the patient may proceed to rapid convalescence, or, after a transient return of warmth, may die in collapse, or he may enter

4.—THE STAGE OF FEBRILE REACTION.—The purging and abdominal cramps subside, the kidneys resume their function, the pulse becomes stronger and the body surface warmer, but in many cases the patient enters a "typhoid state" with high temperature, dry brown tongue, scanty, albuminous urine, apathy or low delirium, from which he may pass to coma and death, or these symptoms after some days may subside and the patient enter a slow, tedious convalescence.

The disease is seen in all grades of severity from the very mild,

walking cases to the most virulent type in which collapse and death occur within a few hours of the onset. *Cholera sicca* is a rare and rapidly fatal form with little or no purging and vomiting.

Complications.—These are apt to appear towards the end of the disease. Pneumonia, nephritis, uremia, diphtheritic colitis, abscesses, local gangrene, parotitis, ulceration of the cornea are all occasionally encountered.

Diagnosis.—The positive diagnosis of sporadic cases can hardly be made except by a careful bacteriological examination of the discharges. The rice-water stools, the rapid emaciation and collapse and the severe muscular cramps are the most characteristic symptoms, but none of these is pathognomonic of the disease. The term *cholera nostras* is applied to a form of severe diarrhea occurring in hot weather, which may closely simulate true cholera and may even prove fatal. It lacks, however, the presence of the specific comma bacillus.

Acute dysentery; the algid form of pernicious malarial fever, and poisoning by mushrooms, ptomaines, arsenic, etc., may be mistaken for Asiatic cholera.

Prognosis.—The mortality of different epidemics varies between 30 and 80 per cent. In general about half of the cases die. The mortality is usually highest at the beginning of an epidemic. Alcoholism, old age, pregnancy, nephritis, debility, etc., are unfavorable features.

Treatment.—Prophylaxis.—In general this is similar to that described for typhoid fever: protection of the water supply from contamination by cholera discharges; the isolation of patients; careful disinfection of all excreta, clothing, bed linen, etc.; the boiling of drinking water and milk, and the avoidance of everything that will tend to derange the gastro-intestinal tract or to lower the general health. Protective inoculation of attenuated cultures of the cholera bacillus have been used with some success by Haffkine in India.

Treatment of the Attack.—The patient is put to bed at once and kept absolutely quiet. The use of a free purge at the beginning of the disease has not been found of advantage. The purging, vomiting and pain are best controlled by the hypodermic use of morphine or by chlorodyne. No attempt at feeding should be made during the active stage. Thirst is relieved by bits of ice or by small quantities of hot water. The enormous loss of body fluid is best replaced by the intravenous or subcutaneous injection of large quantities of normal (0.8 per cent.) salt solution. The surface heat should be maintained by blankets, hot water bags, etc., or by hot baths. Cardiac stimulants are usually needed and should be given hypodermatically in the form of strychnine, digitalis, caffeine, ether, etc. After the active symptoms have subsided feeding should be begun most cautiously by giving fluids in minute quantities at frequent intervals. Indeed, throughout the whole convalescence the greatest possible care in the feeding is required to prevent a return of the purging and vomiting.

DYSENTERY

Definition.—The term dysentery is applied to inflammatory processes of the large intestine, of varied infectious nature, characterized clinically by the frequent passage of scanty, mucous and bloody stools and usually by tormina and rectal tenesmus.

Etiology.—Dysentery has been known for many centuries as one of the great epidemic diseases. It is scattered widely throughout the world, but is most commonly seen, and is most severe, in tropical countries. It prevails chiefly in summer and autumn and attacks all ages and races. It is the scourge of armies in the field and is met with in barracks, jails, asylums, etc. It occurs endemically, epidemically and sporadically. Infection takes place through the gastro-intestinal tract and usually by means of contaminated drinking water. Exposure, fatigue, poor food and slight gastro-intestinal derangements are potent predisposing causes.

The classification is still confused and unsettled. The present status of this subject can be understood only by remembering that until a few years ago all cases of dysentery were classified on the basis of the anatomical lesion, as catarrhal, ulcerative, croupous, etc. The causative relation of the ameba to certain forms, especially of chronic dysentery, was first discovered and recognized in the description of amebic dysentery; later the part of the Shiga bacillus was developed, and called for still further changes in classification. When the causative agent, ameba or bacillus, can be found, we denominate the disease accordingly; in the absence of such knowledge we must fall back on the old anatomical classification. Five types of the disease are usually described. (1) Acute Catarrhal, (2) Acute Specific, (3) Amebic or Tropical, (4) Diphtheritic and (5) Chronic Dysentery.

Acute Catarrhal Dysentery.—This is the mildest form and that commonly met with in temperate climates, where it occurs sporadically and in small local epidemics. It is probable that no single microorganism is responsible for this type of dysentery. Many cases which formerly fell into this class are now known to be due to the *bacillus dysenteriæ* and to belong, therefore, to the type next to be considered.

LESIONS.—There is an acute catarrhal inflammation of the mucosa of the colon and rectum, marked by congestion, increased secretion of mucus, excessive desquamation of the epithelium and hyperplasia of the lymph follicles. Occasionally small points of ulceration cap these swollen follicles.

SYMPTOMS.—The onset is marked by a diarrhea which is at first fecal, but which, within a day or two, assumes a dysenteric character. A chill is uncommon. The painless, fecal movements soon change to small, mucous, bloody passages containing little or no fecal matter. At the same time there are usually severe colicky, abdominal pains (tormina) and distressing rectal tenesmus, with constant desire for stool. The temperature is usually moderately elevated, and there are increasing pros-

tration and loss of weight. The tongue becomes dry and red; the abdomen is retracted and often tender. Vomiting may occur. At the end of a week or ten days, in most cases, the symptoms subside, the stools regain their fecal character and a rapid convalescence is made.

Of the more severe cases a few are rapidly fatal, but most of them run a course of three or four weeks and may then terminate in recovery, in death or in chronic dysentery.

Complications, such as abscess of the liver or intestinal perforation, are uncommon in this type.

Acute Specific Dysentery.—Very recently it has been shown that many epidemics both in the tropics and in temperate regions are due to a specific pathogenic micro-organism—the *Bacillus dysenteriæ*—discovered by Shiga in 1898 in the epidemic dysentery of Japan. This germ has since been found in a number of small epidemics and in many sporadic cases of dysentery both among children and in adults in this country and elsewhere. The organism belongs to the colon-typhoid group of bacilli, is found abundantly in the stools and intestinal contents and is to be identified by certain cultural peculiarities. The blood of patients suffering from specific dysentery, even when highly diluted, agglutinates fresh cultures of this bacillus and this fact has been used as an aid to diagnosis similar to the Widal test in typhoid fever. Its diagnostic value, however, has not yet been fully determined.

The LESIONS of specific dysentery are in no wise characteristic and vary greatly both in extent and severity. The rectum and sigmoid flexure are the regions most commonly affected. Sometimes the whole large gut is involved and the process may even extend to the ileum. In the mildest cases the lesions are similar to those of catarrhal dysentery. More commonly, however, the inflammatory process is more severe and is not confined to the mucous coat. The tissues are infiltrated with serum, leukocytes and blood; a grayish *pseudo-membrane,* composed chiefly of fibrin and the necrosed superficial layers of epithelium, coats the inner surface of the gut and this in certain places may separate by sloughing and leave *ulcers* of varying depth and extent.

The SYMPTOMS are similar in kind to those of acute catarrhal dysentery, but are apt to be more severe. The onset is often violent, the fever high and the prostration extreme. Delirium and coma vigil are present in many of the worst cases. The course varies between a few days and several weeks. The mortality differs much in different epidemics and is apt to be specially high in the tropics, in crowded institutions and among children. When ulceration is extensive the case is apt to be prolonged indefinitely as chronic dysentery. Such complications as liver abscess, perforation, peritonitis and septicemia, are occasionally met with.

Amebic or Tropical Dysentery.—A type of dysentery common in the tropics, and met with occasionally in this country and in other temperate regions, is that constantly associated with the presence in the intestines of an ameba which is believed to be its specific cause. This

Ameba dysenteriæ is a large oval, unicellular organism with active ameboid movements and phagocytic action. It is found in great numbers in the freshly passed mucous stools and is present also in the inflamed intestinal walls and in the pus and walls of the commonly associated liver abscess. The amebæ are best detected by their ameboid movement. To this end the mucous parts of the stool should be placed on a warmed slide and examined microscopically immediately after being passed.

The LESIONS of amebic dysentery are quite characteristic and differ much from those of other types. The essential change is a peculiar gelatinous infiltration and transformation of the *submucosa* with *ulceration* of the overlying mucous membrane. The lesions are confined usually to the colon and rectum. The ulcers are of various shapes, with ragged and undermined edges. The changes in the submucosa are usually much more widespread and severe than the extent of the ulceration would indicate. In long-standing cases there is often much connective tissue proliferation which may result in great thickening of the wall of the gut and narrowing of its lumen. A secondary lesion of much importance is that of abscess of the liver. The abscess is usually, but not always, single. The material from such abscesses consists chiefly of broken-down liver cells, fat droplets, granular detritus and amebæ, with little or no true pus.

SYMPTOMS.—The course of the disease is usually subacute or chronic, although the onset may be sudden and the symptoms at first severe. There are prone to be periods of exacerbation and remission. Pain and tenesmus are less constant than in the other types of dysentery and the stools are by no means always mucous and bloody. The temperature is usually not much elevated, but there are progressive weakness and emaciation. In the chronic cases periods of constipation alternate with those of diarrhea. Abscess of the liver is a common and grave complication. Intestinal perforation is sometimes met with. Death may occur from the severity of the early symptoms, from exhaustion or from abscess of the liver.

Diphtheritic Dysentery.—This term is applied to cases of colitis in which the inflammation is of diphtheritic or pseudo-membranous type. Some of these cases no doubt belong in the class of acute specific dysentery. The whole, or a considerable part, of the large intestine is the seat of a severe exudative inflammation with the formation of a necrotic pseudo-membrane which, in places, sloughs off and leaves areas of ulceration. In the severest cases the whole inner surface of the colon is a black, stinking, gangrenous mass.

PRIMARY cases usually run a severe course and are very fatal. The dysenteric symptoms are often obscured by the severity of the constitutional symptoms and the condition is frequently mistaken for typhoid or for septicemia.

SECONDARY cases are occasionally met with in lobar pneumonia and in the chronic and wasting diseases, such as chronic nephritis, chronic

heart disease, tuberculosis, etc. The condition is frequently overlooked; the symptoms being masked by those of the primary disease.

Chronic Dysentery.—This may follow any of the above described forms. Ulceration, scarring, thickening of the submucous and muscular coats and polypoid outgrowths of the mucosa are the common lesions. The intestinal symptoms vary much from time to time. Diarrhea alternates with constipation and there is usually great loss of flesh and strength. The course of the disease is very long and tedious. A complete cure is difficult to obtain and many cases eventually die of exhaustion and malnutrition.

Treatment of Dysentery.—In all acute cases absolute rest in bed is required. If seen at the outset the patient should be given a purgative dose of Rochelle salts or castor oil. It is essential that the inflamed colon be given as nearly complete rest as possible. With this in view the food should be such as will be easily digested and will leave as little irritating residue as possible. Milk, whey, egg-water or broths should be given in small amounts and at short intervals. The pain and tenesmus are best controlled by the subcutaneous use of morphine or by rectal injections of starch-water and laudanum. Hot poultices or stupes to the abdomen are often grateful. The use in acute cases of astringent or antiseptic drugs internally is of somewhat doubtful value. Bismuth in very large doses ($\mathfrak{z}$ss–$\mathfrak{z}$j per diem) is often useful. In the tropics ipecacuanha, in a single very large dose (gr. xxx), has long been a favorite remedy. In the subacute and chronic cases irrigation of the colon with large quantities of a weak astringent solution (nitrate of silver, gr. xv–xxx to a pint) is often of great value. In amebic dysentery colon irrigations of a solution of quinine (1 to 5000 to 1 to 1000) are recommended. In chronic dysentery care must be taken that strength is maintained by a diet which is nutritious as well as bland and unirritating. In cases hopelessly chronic the operation of right inguinal colostomy is justifiable and has sometimes resulted in complete cure. Irrigation of the colon through the appendix, which has been brought up into an abdominal incision and then opened (appendicostomy) has also been practiced with success. Finally complete removal of the colon has been done in a few cases. In acute specific dysentery the serum of animals immunized to bacillus dysenteriæ has already been used with some measure of success.

APPENDICITIS

Definition.—An inflammation of the vermiform appendix.

Etiology.—The disease occurs at all periods, but is most common between the ages of 10 and 30. It is uncommon but not unknown in infancy and old age. Males are affected two or three times as often as females.

Local conditions of the appendix are important. (a) Conditions favoring the retention of fecal contents, such as stenosis of the mouth

of the appendix, either of the so-called valve of Gerlach or the adjacent portion of the tube. (b) Fecal concretions or foreign bodies of any kind. Parasites, particularly the oxyuris vermicularis, pins and fecal concretions are not infrequently found in the appendix. These foreign bodies, by injury of the mucous membrane, appear to open the way for the invasion of the micro-organisms present.

BACTERIOLOGY.—The infection is nearly always mixed, the bacillus coli being found with other organisms: streptococci or staphylococci, bacillus lactis aërogenes, bacillus aërogenes capsulatus, bacillus pyocyaneus and various other organisms of those which may be found in the intestine.

Morbid Anatomy.—ACUTE APPENDICITIS.—The anatomical changes vary from the mildest type of inflammation (catarrhal) to the severest forms (suppurative or gangrenous). In the milder types the changes may be very slight, simply congestion and slight swelling of the several coats, in the latter the whole appendix may be destroyed with the development of an abscess or a general suppurative peritonitis. The inflammatory changes affect the contents of the appendix, the walls of the appendix, and the surrounding tissues.

Contents.—These may consist of normal feces, of a fecal concretion, a foreign body, or of an inflammatory exudate comprising mucus, or blood, or pus, of varying consistency. The exudate, especially if the mouth of the appendix be closed as the result of deformity or of the inflammatory changes, may distend the appendix sufficiently to aid in or cause its rupture. In severe cases the contents of the appendix have the foul odor usually found in colon bacillus infections.

Walls of the Appendix.—The coats of the appendix may be slightly thickened by the exudate or they may be considerably increased in depth. Often more or less necrosis of tissue is present at a localized area, producing an opening (perforation) through which the contents escape, or a widespread destruction of the organ, so that at operation only the tip or the stump or no part of it may be discoverable. About the area of perforation the adjacent tissue is commonly black, necrotic and foul-smelling.

Surrounding Tissues.—The peritoneal coat of the appendix may show no changes in the very mild cases, or it may be simply congested, or it is the seat of an acute fibrinous, suppurative, or gangrenous inflammation. Corresponding changes occur in the abdominal peritoneum and it may be simply congested or the seat of a general suppurative or gangrenous inflammation. If, however, as often happens, the appendix is shut off by adhesions, an abscess cavity forms about the appendix (localized suppurative peritonitis) containing pus, possibly blood and the remains of the appendix.

Suppurative inflammation, either localized or general, usually results from perforation of the appendix, but may develop without definite perforation, the infecting organisms making their way through the inflamed wall of the organ. The abscess may lie in the pelvis, in the right

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iliac fossa, in the loin behind the cecum or colon, or indeed in any part
of the abdominal cavity.

Remote Lesions.—The suppurative inflammation may produce throm-
bosis of radicles of the portal vein. If these break down secondary
pylephlebitis or multiple pyemic abscesses of the liver may result or
an abscess may form retroperitoneally (subphrenic, etc.). Pulmonary
infarction or thrombosis occasionally follows appendicitis, seemingly
from the dislodgment of a local thrombus in the appendix area.

CHRONIC APPENDICITIS.—As the result of one or more attacks of acute
inflammation, especially of the mild character usually termed catarrhal,
the walls of the appendix may become very much thickened, its lumen
narrowed and distorted and the organ surrounded by dense adhesions.
In some instances the canal of the appendix is obliterated and the ap-
pendix is reduced to a dense mass of fibrous tissue (obliterative appen-
dicitis). To these results of repeated attacks of acute inflammation
which often constitute a more or less chronic process, the term chronic
appendicitis is applied.

Symptoms.—The symptoms of acute appendicitis must vary with
the severity of the attack. In any event they are both general and local.
Pain, usually severe, referred at first to the epigastrium or the abdomen
generally and after an hour or two located in the right iliac fossa, or
over the situation of the appendix, if it be situated elsewhere (the right
hypochondrium or the left side of the abdomen), marks the onset. With
the pain, nausea, vomiting, a rising temperature (102°–103°) and con-
stipation are regularly associated. In rare instances diarrhea occurs.
The prostration varies with the degree of fever and the severity of the
pain, but profound prostration may accompany gangrenous inflammation
with little fever.

PHYSICAL SIGNS.—At the onset there is indefinite abdominal tender-
ness, later tenderness to pressure in the right iliac fossa, often sharply
localized over the base of the appendix at a point mid-way between
the umbilicus and the anterior superior spine (McBurney's point), with
definite rigidity of the muscles of the abdominal wall of the right lower
quadrant of the abdomen. If the appendix is abnormally situated the
local signs are accordingly misplaced, so that they may be found at
any part of the abdomen.

COURSE AND LATER SIGNS.—1. *Resolution.*—In the mild cases the
pain subsides after 24 or 48 hours, the temperature falls, the pulse
returns to normal, and the local signs disappear. In severer cases the
fever, nausea, vomiting, and pain persist, the prostration is marked, the
local signs continue, become more and more marked and the condition
terminates in a localized abscess about the appendix or in acute general
peritonitis, or resolves after several days' severe illness.

Abscess Formation.—More or less local peritonitis commonly accom-
panies appendicitis. In acute inflammation, if the local peritonitis walls
off the appendix from the general peritoneal cavity and the inflammation
continues, a peri-appendicular abscess is produced. Fever continues as

a rule, although an abscess may develop with a falling temperature, and the temperature runs the irregular course of septic inflammation.

The pulse remains more or less rapid, the prostration continues, vomiting may be repeated, sweating may occur, and in protracted cases the patient loses flesh and becomes anemic. The local signs become more and more marked, the tenderness and rigidity persist and resistance increases in the right iliac fossa, and after a time dulness and a definite tumor may be made out in this region. Fluctuation can rarely be determined. If the abscess be pelvic, the right iliac fossa may present little or no abnormality, and the pain, tenderness and tumor may be detected only by rectal examination. Similarly the signs of abscess may develop in the hypochondrium or other part of the abdomen. If opened and drained, the temperature gradually falls, the abscess closes after two or three weeks, and recovery ensues. Unopened, such abscesses may possibly rupture through the abdominal wall, in the thigh below Poupart's ligament or in the loin, but such results are not seen under modern surgical practice. Acute general peritonitis may develop from rupture or extension of inflammation from such an abscess.

Acute General Peritonitis.—This usually results from the severer types of infection and inflammation, but it may follow apparently mild attacks. Its development is usually gradual and marked by steadily progressive increase in both constitutional and local symptoms. It may, however, develop suddenly and unexpectedly in any case of appendicitis. With the onset of acute general peritonitis the fever continues and usually rises, the pulse becomes more rapid, the vomiting more frequent, the prostration more profound, so that the facies becomes ''pinched '' and often ashen. Locally the abdomen becomes distended, painful, tender, and rigid in all parts, and the illness runs the course of acute general peritonitis from any cause (see page 143). Unless relieved by operation death is inevitable within a few days.

Special Symptoms.—Head's zones—cutaneous hyperalgesia. Hypersensitiveness of certain definite areas of the skin has been found to be associated with disease of the viscera. The areas thus affected are definite and fairly fixed and their demonstration is helpful in the recognition and differentiation of visceral affections. The cutaneous hyperalgesia is demonstrated by pinching the skin or lightly and evenly stroking it with the point of a needle, first from side to side and then from above downward, the patient reporting whenever the sensation becomes painful and the point thus indicated being marked. In the case of appendicitis the area thus outlined is found to cover the right half of the abdomen below the level of the umbilicus and not quite reaching Poupart's ligament, an area corresponding to the right 10th and 11th spinal segments.

The Blood.—A definite leukocytosis is regularly present, 12,000 to 15,000 or more, and in the severest cases the count often exceeds 30,000. Similarly there is an increase in the percentage of polynuclears above the normal of 75 per cent., and sometimes amounting to 90 per cent. to

95 per cent. of the total amount. In a general way these two reactions are associated, the higher the leukocytosis, the more likely the polynuclear percentage is to be increased, and as a rule they correspond to the severity of the attack. Thus the mild attacks show little or no leukocytosis and a normal polynuclear percentage; the severe cases usually show high total counts and high polynuclear percentages. Many exceptions to these rules are observed, and it is quite possible that an acute appendicitis with perforation of the appendix and beginning acute peritonitis should show a low leukocyte count and no disturbance of the ratios. The blood reaction should be regarded as one of the symptoms of the disease and valued accordingly.

Chronic Appendicitis.—Two types are recognized.

1. Persistent local tenderness and constipation with more or less intestinal indigestion, shown by nausea, tympanitic distention and a coated tongue, mark the first type.

2. The second type is marked by recurrent brief attacks of pain, tenderness and rigidity in the right iliac fossa without constitutional disturbance, or by frank attacks of acute appendicitis. Any one of these attacks may develop into severe appendicitis with abscess formation or general peritonitis as a sequel. These cases are frequently mistaken for gastric ulcer or for gall-stones.

Diagnosis.—An acute inflammatory condition of any of the abdominal viscera may give the same general symptoms as acute appendicitis, especially affections of the stomach, gall-bladder, right kidney, and the tube and ovary of the right side. For differentiation we must depend largely upon a knowledge of a cause for such disease, of previous disease of one of these organs, the location of the pain and physical signs, and the relative frequency of acute appendicitis. Attacks of acute appendicitis, moreover, regularly occur without apparent exciting cause.

Perforating gastric or duodenal ulcer gives a history of previous gastric indigestion, or possibly hematemesis, and the local signs are found in the epigastrium or the left hypochondrium.

Acute cholecystitis or cholangitis follows gall-stones or jaundice or develops in the course of typhoid fever or pneumonia, and the local signs center in the right hypochondrium adjacent to the 9th costal cartilage.

Acute pyelitis or abscess of the kidney is indicated by previous history of genito-urinary infection, urethritis or cystitis, by the presence of pus or blood in the urine and local tenderness in the flank as well as in the abdomen.

Renal or uteral (right) colic may closely simulate acute appendicitis, but the presence of blood in the urine and the results of X-ray examination are decisive.

Dietl's Crises, due to floating kidney, should be recognized by the demonstration of the presence of a movable organ.

In women acute inflammations of the right appendages (tubes and ovaries) are often mistaken for appendicitis. A history of previous pel-

vic disease (vaginitis or endometritis, pelvic cellulitis) is helpful and the location of the local pain, tenderness and rigidity in the groin and the results of vaginal examination are important.

Acute enteritis arises from definite cause, overfeeding or improper food, as a rule, is accompanied by little fever, and free diarrhea, and the local signs are centered about the umbilicus.

Mucous colitis gives rise to attacks of pain, fever and constipation, easily mistaken for acute appendicitis. The disease belongs especially to neurotic women and a history of previous attacks with the passage of large quantities of mucus can be obtained.

Typhoid fever may begin with such acute abdominal symptoms as to suggest appendicitis. The history of several days' illness before coming under observation, the headache, insomnia and prostration, diarrhea if present, and the low leukocyte count, the enlargement of the spleen, and possibly the roseola should indicate the correct diagnosis.

Pneumonia, especially in the young, may begin with abdominal pain, tenderness and rigidity, very suggestive of appendicitis. Very high fever, rapid respiration, cough or expectoration should excite suspicion, and careful physical examination will usually elicit signs of dry pleurisy or consolidation. Several days' observation may be required to settle the question.

Prognosis.—While it is true that even the mildest cases of appendicitis may be complicated by serious conditions, even general peritonitis, as a rule those with mild symptoms at the outset remain mild and recover promptly. In the severer cases prognosis is always difficult because of the dangers of localized peritonitis or abscess, or acute general peritonitis, and the possibility of later disturbances. The mortality of cases of appendicitis not operated upon lies between 5 and 10 per cent.

Treatment.—Absolute rest and quiet are essential. The patient is put to bed, the bowels emptied by a high enema or by a mild purgative, such as castor oil or calomel. Many object to the use of purgatives in appendicitis, but their reasonable use does no harm. All feeding should be stopped and only water or cracked ice allowed by mouth. For the relief of pain heat by the hot water bottle, poultice or hot fomentations, or an ice-cap may be employed. Opium or morphine must be employed if the pain is intense, but should be given in small doses, as undoubtedly large doses of opiates benumb the patient's senses and mislead the physician as to the true conditions.

Mild cases improve promptly and usually are well in a few days. Feeding is resumed as the symptoms subside.

Immediate surgical intervention is advised by some for all cases, and is certainly to be recommended for all beginning severely or becoming more severe after the onset. Surgical advice should therefore be had in all such conditions. As a rule the early operations are the safest, and only under special conditions is operation delayed, when deemed necessary. Even when acute general peritonitis has developed many of the patients are saved by surgical treatment.

INTESTINAL OBSTRUCTION

(Ileus)

Etiology.—Intestinal obstruction is produced by a variety of mechanical conditions which block the intestine and stop the normal progress of the contents. The symptoms of obstruction are explained first by the failure of the normal evacuations (constipation, more or less absolute), and secondly by a reverse peristalsis which develops in the portion of the intestine above the block and empties it by discharging the contents into the stomach, from which they are ejected by vomiting.

The causes of obstruction may be classified thus:

1. Strangulation, the constriction of the bowel in one of the many hernial pockets of the abdomen, or by bands or cords, such as peritoneal adhesions, Meckel's diverticulum, the remains of the vitelline duct, or an appendix adherent by its tip alone. The numerous fossæ formed by the peritoneum (twelve are described) may, any of them, be the site of strangulation. One-third of all cases of obstruction are produced in this manner. Males are more often affected, especially in the years from 15 to 30. The obstruction is nearly always in the small intestine.

2. Invagination, the telescoping of one part of the intestine into another. One part of the intestine becomes unusually contracted by excessive peristalsis and the portion below is then drawn upward over it. The process regularly begins at or near the ileocecal valve, and the invaginated portion may be carried downward through the colon and rectum till it presents at the anus. (See Fig. 19.)

Invagination occurs especially in infants under one year, but may occur in children up to the tenth year. It is unknown in later life. This process also accounts for about one-third of all cases of obstruction.

As a result of invagination a tumor is formed, composed of the two tubes of intestine, one inside of the other, and the mesentery of the telescoped portion. The latter portion is spoken of as the intussusceptum, the outer tube as the intussuscipiens. This included part becomes intensely congested and hemorrhages of some amount occur from the engorged mucous membrane. Recovery may occur normally either by release of the intussusceptum and return to natural relations, or by the sloughing off of it with union of the intestinal walls at its base. Fibrous stricture at the point of juncture may result.

3. Twists (volvulus) and Knots.—These occur in nearly all cases in the sigmoid flexure, near the rectum. The bowel may be twisted on its long axis, a half or whole turn, producing complete strangulation. About 14 per cent. of cases of obstruction are thus explained. This condition belongs to adults, 30 to 40 years of age, especially men.

4. Strictures and Tumors.—Congenital stricture by malformation, such as imperforate anus, is well known. In later life strictures result from the healing of ulcers, syphilitic, stercoral, or tubercular, dysenteric, or typhoid, or from localized peritonitis. Tumors of the intestine

may obstruct its lumen by growth, or the obstruction may be caused by the pressure of tumors in other organs, such as uterine fibroids.

Cancer, especially in women over 40, is the most common, but all manner of tumors have been found. Among the rare forms to be mentioned are papilloma, fibroma, adenoma, or lipoma.

5. Abnormal Contents.—Gall-stones and fecal impaction are the common forms. Gall-stones are seen especially in women over 50. Fecal impaction may occur in either sex at any age. Enteroliths may be

Fig. 19 —An ileocolic intussusception. Small intestine above; colon below. Apex of intussusception swollen, congested and covered with membranous exudate. Appendix in upper left-hand corner.

formed with bits of bone, masses of hair or other foreign substance as a nucleus. They may have become large enough to obstruct the bowel. Foreign bodies may be swallowed or introduced into the rectum and eventually give rise to obstruction, although the great majority are regularly passed without harm.

Peritonitis.—In any form of intestinal obstruction a localized peritonitis is likely to develop at the site of the obstruction. Especially in strangulation, intussusception and volvulus, in which gangrene of the

bowel occurs, the peritonitis may become general and the clinical picture gradually changes to that of acute general peritonitis.

Symptoms.—These include (1) pain, (2) constipation, more or less absolute, (3) vomiting, persistent, (4) tympanites, and possibly (5) tumor.

The PAIN is at first colicky, later continuous. It may be referred to the seat of obstruction or be general. Localized tenderness may be present, but is more often not observed.

CONSTIPATION may be marked from the beginning or the contents of the bowel below the obstruction may be passed at first with no subsequent evacuations. In intussusception, however, the engorged intussusceptum may discharge blood and mucus, which are passed in frequent small stools, at times suggesting dysentery. In complete obstruction of the bowel not even gas may be passed by rectum. The VOMITING is produced by reverse peristalsis, emptying the bowel, so we see in succession stomach contents, bile-stained (green) fluids from the duodenum, then the contents of the lower intestine which gradually assume the character and odor of fluid feces. These changes in the nature of the vomited material may be shown in the course of 24 hours, but the so-called fecal vomiting is usually seen at the end of the second or third day.

TYMPANITES is produced by the distention of the portion of the bowel above the obstruction. It is more marked the lower the site of the lesion. The pattern of the distended intestines shown through the abdominal wall may therefore be of help in determining this point. If the obstruction is in the upper part of the intestinal tract there may be little distention, or the tympanitic area may be limited to the stomach and adjacent portion of the duodenum.

A palpable TUMOR is most often found in cases of intussusception. It then has a very suggestive sausage shape and feeling. Tumors may also be met with in the case of new growths either in the wall of the intestine or, in cases where the obstruction is produced by pressure from without, in other abdominal viscera. As already noted, in intussusception the tumor may also be felt by rectal examination. Fecal impactions may be made out by either abdominal palpation or rectal examination, or both.

CONSTITUTIONAL SYMPTOMS are usually slight at first but rapidly become marked. Fever is absent at the onset, but after the first day the temperature either shows a slight rise or becomes subnormal. It may remain below normal throughout. The advent of peritonitis is marked by a rise of temperature and characteristic local signs. Owing to the persistent vomiting there is great thirst, and the urine is scanty and high-colored. The urine may show albumin and casts and may contain much indican. Prostration is regularly marked from the beginning. The pulse, at first but little affected, regularly becomes rapid and weak. Unless the obstruction is relieved the patient succumbs from exhaustion or from peritonitis in from three to six days.

CHRONIC OBSTRUCTION is most often seen as the result of stricture, either simple or malignant, or fecal impaction. In these cases there is a history of repeated attacks of obstinate constipation with abdominal pain and distention and nausea or vomiting, each attack becoming severer than the preceding one and being relieved with greater difficulty, till one ends in the picture of complete and permanent obstruction.

In cases of fecal impaction the bowel may be enormously distended, its wall eroded and even perforated without the production of complete obstruction. After a period of constipation there may be diarrhea, the fecal mass in some cases being channelled so as to permit the passage of fluid material from above. The fecal tumor may be located by abdominal examination or exploration of the rectum. It is usually of large size, has a characteristic doughy feeling and can be indented by the fingers, and is painless.

With simple or malignant stricture there is the same history of increasing difficulty in securing evacuation of the bowels, with attacks of partial obstruction from time to time. Finally the obstruction becomes complete and the symptoms are those of acute obstruction, but the progress of the affection is usually slower than in the other types of that condition. In elderly people with malignant stricture there may be emaciation and anemia, but it is not uncommon to see carcinoma produce local obstruction without constitutional symptoms.

Diagnosis.—The onset of the symptoms enumerated without fever, and the gradual change in the nature of the vomitus are characteristic of intestinal obstruction. We must, however, exclude the presence of certain acute inflammatory processes which may give a similar picture. Appendicitis is differentiated by early fever and rapid pulse, and the local pain, tenderness and rigidity. The bowels respond to enemata. Peritonitis also begins with febrile symptoms, general and exquisite pain, tenderness and rigidity. The vomiting of peritonitis resembles that of obstruction, but never becomes truly fecal. Pancreatitis (acute hemorrhagic) may readily be mistaken for intestinal obstruction, but is usually recognizable by early febrile symptoms, the extreme pain, tenderness, distention and rigidity in the epigastrium without the vomitus characteristic of obstruction. Having excluded these possibilities we must seek to determine the site and nature of the obstruction.

THE SITE.—The higher in the intestinal tract the obstruction occurs, the earlier and more severe the vomiting, and the earlier will collapse appear, and the less the general abdominal distention. The lower the location of the obstruction, the slower the evolution of the symptoms and the more marked the distention. The outlines of distended intestines may indicate the site of obstruction. Thus in low obstruction the colon will stand out prominently. With the lesion near the ileocecal valve, the distention is mainly about the umbilicus and the colon is flat. With high obstruction only, the stomach and adjacent parts of the intestines will be distended. The location of a tumor or mass in the abdomen or pelvis may be conclusive as to this point.

NATURE OF OBSTRUCTION.—The age and sex of the patient are important, as already indicated. In children intussusception is by all means the most frequent form, in adults strangulation. All accessible sites of hernia must be carefully examined, remembering that we have no means of detecting hernia into any of the peritoneal fossæ mentioned above. Knowledge of a previous peritonitis or previous abdominal operation, either of which may leave adhesions, is helpful. Such conditions are naturally most frequently met with in women. In men over forty volvulus must be thought of, while in women of like age gallstones are common. At this time malignant disease is possible in either. Fecal impaction may be found at any period. Finally the exact nature and site of the lesion can often be determined only by operation.

THE BLOOD.—A leukocytosis is regularly present in acute obstruction. In some cases the count shows 50,000 or even 80,000 leukocytes to the cubic millimeter.

Prognosis.—Acute intestinal obstruction is nearly always fatal unless promptly relieved. Children will occasionally recover from intussusception without operation, but the possibility is too slight to be of value. Obstruction from gall-stones or fecal impaction may also yield naturally, but the attacks will be repeated unless the cause is removed.

Treatment.—In any case of suspected acute obstruction feeding should be stopped. Purgatives must not be given. The persistent vomiting may be relieved by repeated washing of the stomach. The bowel below the obstruction may be emptied by enemata of warm water, soap-suds or olive oil. In giving injections the amount of fluid retained should be carefully measured as an indication of the patency of the colon. In adults the capacity of the colon may amount to six quarts. For the relief of pain morphine may be given hypodermatically. For the distention turpentine stupes to the abdomen are indicated. Intussusception in children may sometimes be relieved by the injection of water or air into the rectum and colon. For this purpose the child is anesthetized, placed upon an inclined plane with the head downward, and water is then allowed to flow from a fountain syringe, suspended not more than three feet above the level of the table, into the colon. The passage of the water upward in the colon may be followed by observation of the abdomen, and the reduction of the intussusception may be favored by gentle manipulation of the tumor, if it can be felt. Instead of the water air may be injected from a Davidson's syringe. These measures are applicable only in the early stages of intussusception (the first 24 hours) and are always attended with some risk of rupture of the weakened intestine. In the event of their failure or of the return of symptoms after temporary disappearance, the abdomen should be promptly opened and the intussusception reduced by traction and manipulation. In all other cases of acute obstruction operation is imperative as soon as the diagnosis is made. In cases of chronic obstruction the diet should be restricted, the bowels moved by enemata, and operation undertaken as soon as the permanency of the obstruction is established.

CONSTIPATION

Etiology.—Normal defecation is the result of the co-ordination of a number of factors. Feces accumulating in the rectum and colon excite the mucous membrane from which nerve impulses travel to a center in the lumbar cord and there cause motor impulses which result in increased peristalsis of the intestine and a simultaneous relaxation of the sphincter ani. The cerebrum exercises a certain degree of control over the lower centers in the cord and may thereby inhibit or augment their action. Furthermore, in the final operation the voluntary muscles of the abdomen as well as those of respiration are called into action, so that the process can be materially modified by conscious action. Constipation may result from interference with any of these several steps.

1. Modification of the fecal contents by too scanty or otherwise unsuitable food, or by disturbances of digestion in the stomach or intestine.

2. Impaired sensibility of the intestinal wall, as the result of disease or injury—such as occurs in certain forms of colitis, or atrophy of the mucous membrane. The taking of drugs, especially opium or its derivatives, probably operates in this way.

3. Impairment of any part of the reflex nervous mechanism by which the sensory impulse is translated into motor discharges, such as occurs in many nervous diseases, particularly those affecting the cord.

4. Inattention to or inhibition of the natural impulses to defecation. Infants over six months of age can be trained to have a defecation at a regular hour. In children particularly, but often in adults as well, the same influence of habit can be seen. Constipation may be said to be a normal condition in idiots and the insane.

5. Lack of muscular power in the colon or in the voluntary muscles which take part in the process. Loss of power in the colon is seen in many cases of chronic inflammation of the colon, in hysteria and neurasthenia. In conditions of relaxation of the abdominal muscles, such as follows pregnancy, constipation results.

6. Lack of normal exercise affecting these muscles. Constipation is almost unknown in workmen whose occupations call for abundant use of the abdominal and respiratory muscles. It is the rule in men of sedentary life.

7. Abnormalities which interfere with the emptying of the rectum and colon. In children the extreme length of the sigmoid operates in this way. Later in life strictures of all kinds are met with. Tumors of other parts, such as fibroids of the uterus, by pressure upon the intestine, may obstruct it.

8. Spasm of the sphincter ani. This is not an infrequent form of obstruction. Spasm may be the result of excessive nervous irritability, such as is seen in neurasthenia, or it may result from the presence of local conditions, ulcers, fissures, or other painful affections of the anus.

In most cases of constipation several of these factors are combined to bring about the result.

While the conditions under which constipation is met with are manifold, the condition is most closely associated with the acute fevers, chronic diseases of the stomach, intestine or liver, and the various anemias, especially chlorosis.

Constipation in infants may result from congenital abnormalities of structure or function. The long sigmoid flexure is a factor of importance, and congenital strictures, as well as imperforate anus, are known. Most often constipation in infants is the result of improper feeding and lack of training. The habit so developed may last throughout life. Condensed milk and various proprietary foods by reason of deficiency in fat may produce constipation. Anemia, rachitis, and other organic diseases are causes of importance.

Symptoms.—Constipation for long periods may be borne without discomfort by some. It is, however, usually associated with headache, lassitude, loss of appetite, possibly nausea, and malaise. The tongue is coated and the breath offensive. Fever is also present at times. In young children constipation quite regularly gives rise to attacks of fever, fretfulness, and vomiting. In extreme cases the feces form masses in the rectum or colon, which may obstruct the bowels, enormously distend the part in which they lodge and cause enteritis, or even perforation and peritonitis. A doughy tumor which can be indented by the finger is found either in the abdomen or by rectal examination. Hemorrhoids or anal fissures may be caused by the pressure of fecal masses.

Treatment.—In many cases this must be by the removal of the cause, such as the pressure of a tumor, the cure of an anal fissure, the removal of a mass of hardened feces, or the treatment of the associated neurasthenia, hysteria, or anemia, or disturbances of the other digestive organs. At the onset of acute fevers we relieve constipation by a full dose of calomel and salts, or castor oil, or a simple enema. During the course of such fevers the bowels had best be moved by enema.

To treat the habit of constipation properly we must combine: (1) Regularity of habit, a fixed hour for defecation observed daily. (2) Exercise for the abdominal muscles, such as (a) Standing erect and bending to touch the fingers to the floor without bending the knees. (b) Lying flat upon the floor and raising the body by the pelvic muscles without using the hands. This exercise can be made more effective by hooking the toes under some support, as the end of a bed, and swinging forward against the knees as a rower does in rowing. (c) Hooking the thumbs together with arms extended above the head and then swinging as far as possible from side to side. Such exercises should be practiced for 10 to 15 minutes on rising. When exercises cannot be taken, massage to the abdomen may be substituted, or a cannon-ball weighing 4 to 6 pounds may be rolled over the abdomen. (3) Diet, suited to the individual digestion. Concentrated foods, leav-

ing little residue, such as milk and eggs and meat, are undesirable. Oatmeal, graham breads, or brown bread, which leave much residue are helpful. Fruits of all kinds, but especially oranges, figs, dates and apples, are laxative in effect. A glass of water, hot or cold, sipped before breakfast, is of value. Coffee and tobacco help some persons. Drugs are to be used only in the failure of hygienic and dietetic measures. Cascara sagrada is given, 5 to 10 grains of the extract in pill, each night or in smaller doses after each meal. Rhubarb and soda mixture, pills of aloin, belladonna and strychnine, or combinations of these drugs with podophyllin, are often used. Suppositories or enemata may be required. In that case, suppositories of glycerine, or enemata of warm water, soap-suds, or olive oil may be used. In the obstinate constipation of neurasthenia or hysteria, high injections of 1 to 1½ pints of warm olive oil may be the only remedy. Enemata of water or oil may be used for years in some cases without harm.

In infants the measures of most value are (1) increase in the percentage of fat as high as can be borne; (2) the use of oatmeal instead of barley water in the food; (3) the addition of orange juice. A conical piece of soap may be inserted into the anus and held there for a few minutes. Glycerine suppositories may be used. Abdominal massage is of value. If medicines must be used milk of magnesia is best, one or two teaspoonfuls added to the evening feeding. Rhubarb and soda or preparations of cascara sagrada may be used for a time.

ENTEROPTOSIS

(Glenard's Disease)

Definition.—An abnormal downward displacement of the intestines (usually also the other abdominal viscera). Thus we may see gastroptosis, splenoptosis, hepatoptosis, or nephroptosis.

Etiology.—The condition may be congenital or may in later life be due to relaxation of the various abdominal ligaments produced by repeated pregnancies, ascites, or the like. In some cases increased weight of the individual viscera, such as occurs in dilatation of the stomach or enlargement of the spleen or kidney, is the cause. It also develops in certain patients without discoverable cause.

Symptoms.—There may be none, or the displaced viscera, especially the spleen, liver, or kidney, may cause more or less constant dragging pain or discomfort. The latter symptoms are sometimes associated with symptoms of neurasthenia or hysteria, if the patients know of the visceral displacement. Rarely the displaced viscus, spleen, or kidney may cause symptoms by pressure on other abdominal organs, such as the uterus, stomach, or appendix. Chronic obstruction of the intestine may result from kinks or twists.

Treatment.—(1) Abdominal supports of various kinds have been designed to meet the indications. (2) The neurasthenia or hysteria must be treated, and the general health guarded. (3) Rarely operative

replacement or removal of a displaced viscus is called for. (See En-
largement of Spleen and Floating Kidney.)

The use of the X-rays following the ingestion of a gruel or paste
containing an ounce of bismuth subcarbonate most accurately locates the
position of the stomach, and if the course of the bismuth downward be

FIG. 20.—Gastroptosis with dilatation of the stomach. Radiograph after administration of emulsion
of bismuth.

followed the position of the intestines. Obstruction may be revealed
by delay in the onward passage of the bismuth. To determine the posi-
tion of the colon, enemata of bismuth are employed. (See Figs. 20
and 21.)

NEUROSES OF THE INTESTINE
(Nervous Diarrhea)

Definition.—A functional motor disturbance of the intestine
producing diarrhea.

Etiology.—(1) The exciting cause is powerful emotion, such as
grief, anger, or fear. The looseness of the bowels experienced by many
speakers just before or after a public appearance is a good example. (2)
The underlying cause may be malnutrition, anemia, or neurasthenia.

The affection sometimes appears in association with nervous diseases, such as exophthalmic goitre or locomotor ataxia.

Symptoms.—Frequent loose, watery movements of the bowels are characteristic. These may occur at any time of day or follow regularly the taking of food. The excessive peristalsis is often accompanied by audible rumbling or gurgling. The course of the affection may be very

Fig. 21.—Ptosis of the transverse colon. An emulsion of bismuth subcarbonate has been injected per rectum and the patient required to lie for five minutes first upon the left side, then upon the back, then upon the right side. The loops of the colon at the hepatic and splenic plexuses are caused by the ptosis of the transverse colon. This condition is not uncommon and its pathologic importance is doubtful.

brief, limited to the immediate action of the exciting causes. In other instances the underlying conditions are the important ones and the condition persists with periods of improvement until they are relieved.

Treatment.—Release from nervous excitement may be all that is required. In the protracted cases we must treat the underlying condition rather than the diarrhea. To this end change of scene and air may contribute. In extreme case the rest cure may be required. Limitation of the diet to easily digestible foods is indicated. Opiates should not be given and astringents are of no value. Nerve tonics, such as asafetida, valerian, arsenic, or the glycerophosphates, are indicated.

MUCOUS COLITIS
(Membranous Colitis)

Definition.—An affection characterized by attacks of abdominal pain or discomfort and the passage of masses of mucus often in the form of shreds or casts of the bowel.

Etiology.—The disease belongs to the years between 30 and 50, but may develop in younger or older persons. Women, more often than men, are affected. Neurasthenia, hysteria, or the nervous temperament is regularly associated.

It is often secondary to chronic constipation or to chronic appendicitis or dysentery.

Morbid Anatomy.—No definite lesions of the colon have been demonstrated. The bowel may be either dilated or contracted.

The mucus discharged is usually in the form of large colorless or whitish gelatinous masses or shreds or casts of the bowel. It may simply coat the surface of a formed stool or be mixed with fecal material, or constitute the entire passage.

Symptoms.—The onset of the disease is gradual and the process is usually well developed before it is recognized. Attacks of abdominal discomfort, soreness, or severe colic occur from time to time and accompanying or following the attacks of pain, the patient passes stools consisting wholly or in great part of mucus. The mucus may appear in masses, or have the form of a cast of the bowel, the patient often being persuaded that the "lining of the bowel" has been passed. In the intervals between attacks the patient suffers from chronic constipation. Lack of appetite, nausea, heaviness or discomfort after eating, abdominal distention, gaseous eructations, heart-burn and the like symptoms of "nervous dyspepsia" may be complained of. The patients become thin and possibly anemic. Neurasthenia, melancholia, or hysteria is often present in marked degree. The affection is very chronic and may continue indefinitely with periods of remission and exacerbation.

Examination of the abdomen at the time of the attacks shows it either distended or flattened, often very tender to pressure, especially on the right side. On inspection of the bowel through a speculum, it may be found distended or contracted, its surface may appear congested, but usually shows only a coating of mucus. Ulcers or other organic lesions are rarely found.

The patient shows the general characters of hysteria, neurasthenia, or melancholia.

Diagnosis.—The passage of masses, or casts of mucus associated with abdominal pains, digestive disturbances and general nervous manifestations is usually diagnostic. The severe attacks often suggest acute appendicitis, but fever and leukocytosis are absent, and the recurrence of the attacks with the mucous stools diagnostic. Chronic appendicitis can be excluded only with great difficulty, except by the general diffusion of the pains over the abdomen and the absence of focal signs.

Other causes of abdominal pain, such as renal or ureteral stone, disease of the tubes or ovaries, appendicitis and the like, must be excluded.

Prognosis.—Mucous colitis is never fatal of itself, but tends to become chronic and incurable. Prompt and permanent cure may, however, be effected.

Treatment must be adapted to the patient.

The relief of chronic constipation by dietetic and hygienic measures often cures mucous colitis. (Von Noorden.) The institution of a proper diet with exercise and the free use of water may be all that is required. Laxatives should be used only in case of necessity, but some patients are relieved by occasional doses of castor oil. Daily lavage of the colon with water containing the fluid extract of hamamelis (3j to the pint) or nitrate of silver or quinine 1/5000. This should be practiced for a short time only. Injections of olive or cotton seed oil (8–12 ounces) at bedtime are helpful to some.

Abdominal massage or electricity may be used. Neurasthenia or hysteria must be appropriately treated. And finally surgical measures, such as the removal of a chronically inflamed appendix or an appendicostomy to allow free irrigation of the colon from above downward, may be necessary.

CANCER OF THE INTESTINE

Etiology.—Cancer of the intestine, including both large and small intestine, is comparatively common. About 50 per cent. of all cancers are found in the alimentary tract, 8 per cent. of all in the intestine. Most cases occur in the period between 40 and 60 years, a few earlier. Cancer of the rectum is more common in men.

Morbid Anatomy.—Cancer of the intestine is nearly always primary, rarely metastatic. The new growths are of various types. The most common is the cylindrical epithelial growth infiltrating the wall of the intestine; medullary carcinoma, forming soft, sometimes cauliflower-like tumors, readily ulcerating and producing hemorrhages, is not infrequent. Colloid and scirrhous growths are both rare and, if present, are usually in the rectum.

Carcinomatous growths may develop in any part of the intestine, but are most common in the rectum. They form more or less extensive growths infiltrating the wall of the intestine, usually constricting it, showing on the internal surface ulceration of the character seen in cancer of the stomach, and producing by extension into surrounding parts a tumor of varying size. There may be extensive adhesions about the affected part of the gut, partly due to invasion, partly to secondary local peritonitis. If perforation occurs a localized abscess or a general peritonitis is found.

Symptoms.—Cancer of the intestine presents several different clinical pictures.

1. Cases marked by intestinal obstruction. The constriction of the

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intestine may produce obstruction before any other symptoms appear. The obstruction is naturally gradually progressive. The history is therefore of repeated attacks of more or less severe constipation, relieved in one or another manner, terminating in an attack of undoubted intestinal obstruction, with complete obstipation, nausea, fecal vomiting and abdominal pain. On examination the abdomen is found distended, slightly tender, tympanitic. A tumor may be palpable in the abdomen or may be felt per rectum, but in many cases neither the presence nor location of a growth can be made out. Peristaltic waves in the intestine may be observed, and study of these and the location of the abdominal distention (whether of the small intestine, or colon, or both) may suggest the location of the growth.

2. Cases marked by obstruction and ulceration. These are most common in the rectum. Increasing constipation with small, possibly ribbon-like stools, with traces of blood, or muco-pus is the rule. In other cases frequent small passages, attended with straining and containing much pus and blood and little fecal matter, are reported. In either case examination of the rectum by the finger or the proctoscope locates a growth in the rectum, often in the lower part. Pain is usually not severe in the early stages. With ulceration it becomes more and more severe. Cachexia develops relatively late. Fever occurs only in advanced cases.

3. Cases in which the cachexia and possibly fever are present for some time before the growth presents localizing symptoms. In rare cases old people suffering from cancer of the intestine present no evidences but cachexia and an irregular fever.

Course.—Carcinoma of the intestine grows, gradually invading adjacent structures, producing metastases, especially in the liver, and in time causing death either from intestinal obstruction, or pain and cachexia, or such complications as abscess or cystitis caused by localized extension, or general peritonitis. The duration of life after diagnosis varies greatly. Cancers of the rectum are usually discovered early, those of the intestine or colon late. Cases with marked intestinal obstruction are ordinarily promptly fatal. Removal may bring temporary relief. Life in any case is rarely prolonged beyond a year. A few cases of recovery from cancer of the rectum are known.

Diagnosis.—In either acute or chronic intestinal obstruction in a patient beyond 40 years of age the possibility of a new growth of the intestine must be considered. Marked secondary anemia and cachexia strengthen the suspicion. A local tumor of the intestine almost establishes the diagnosis.

Such growths are usually freely movable, but may be fixed by adhesions. They are firm, slightly tender, do not indent on pressure. Fecal impactions must be excluded, remembering that fecal tumors may form above the stricture of a new growth. Repeated enemata or colon irrigation may be employed to aid diagnosis. In many cases the diagnosis can be made only after opening the abdomen, possibly only on microscopic examination of the growth.

Rectal carcinomata, being accessible either by the finger or procto-scope, can as a rule be easily recognized. They form dense cylindrical or polypoid growths infiltrating the wall of the intestine, stenosing it, showing ulceration on the surface, bleeding readily, and movable or fixed, depending upon involvement of adjacent structures. Syphilitic stricture must be excluded, by the history, by the absence of dense infil-tration or deep ulceration, failure of the Wassermann reaction, and the results of treatment.

Tubercular ulceration implies tuberculosis elsewhere, less infiltration and the presence of tubercle bacilli in the mucus of the feces.

If the growth is accessible microscopic examination of fragments taken from the edge of an ulcer may be employed. Rarely fragments of these growths can be found in the feces.

The urine will show indican in abundance. The blood examination indicates severe secondary anemia.

Treatment must be surgical or merely palliative. Removal of the growth, if feasible, should be tried. Exposure to the emanations of radium has been helpful in non-operative cases, especially those of the rectum.

DISEASES OF THE LIVER

JAUNDICE

Definition.—Jaundice is the pigmentation of the skin, conjunctivæ and other tissues or fluids of the body by bile-pigments. Formerly it was taught that jaundice might be produced either by obstruction to the bile-ducts with resulting reabsorption of bile-pigment into the blood or by excessive destruction of red-blood cells, the hemoglobin derived from which was converted into bile-pigment, and jaundice was there-fore classified as (1) obstructive or hepatogenous and (2) non-obstruc-tive or hematogenous. We now know that the latter condition is also due to obstruction to the flow of bile, produced by increased viscidity of the bile and possibly by a catarrhal swelling of the finer bile-ducts in the liver. The latter is therefore designated as toxemic jaundice, and for clinical convenience we restrict the term obstructive jaundice to the cases in which there is obstruction of the larger bile-ducts.

OBSTRUCTIVE JAUNDICE

Etiology.—This may be due to (1) inflammatory swelling of the mucous membrane of the duodenum and common duct; (2) obstruction of the common duct by gall-stones, parasites or other foreign bodies; (3) by pressure from without, especially by tumors of the head of the pancreas, the liver, kidney, stomach or omentum; (4) by tumors growing in the wall of the duct itself; (5) by stricture or obliteration of the duct. Obstruction of the cystic duct does not give rise to jaundice.

Symptoms.—(1) Icterus or the pigmentation of the skin and con-

junctiva. This varies from a faint yellow to a deep olive-green in the permanent obstruction. All the other tissues of the body may be more or less stained. (2) Absence of bile in the stools, giving rise to slate-gray or clay-colored stools. Constipation is regularly present and the stools are hard and dry. (3) Presence of bile in the urine and sweat. The color of the urine becomes darker, with a greenish-yellow tinge. In extreme cases it may be almost black. The sweat may be sufficiently tinged to discolor the linen. (4) Slow pulse: the pulse falls to 40, 30 or even 20 per minute. Frequently the temperature is subnormal at the same time. These effects are attributed to the action of the bile-salts and disappear in chronic jaundice because in the end the liver loses its power to secrete these salts. These symptoms are therefore most characteristic of catarrhal jaundice. (5) Itching of the skin is a common and sometimes very annoying feature of jaundice. (6) Hemorrhagic tendency: in protracted jaundice the coagulation time of the blood may be ten to twelve minutes instead of the normal of three or four. Bleeding may occur from the mucous membranes or into the skin. This tendency to bleed magnifies greatly the danger of operations upon these patients. (7) Cerebral symptoms: headache, lethargy, and melancholia, are characteristic of the early stages of jaundice. In the later stages and in the severer forms, delirium, coma and convulsions may develop. Frequently with these symptoms the patients have an increasing fever, dry tongue, a rapid pulse and all the other symptoms of a severe intoxication. This condition is spoken of as cholemia, as though produced by the bile-salts, but at present there is no good evidence that such is the case.

TOXEMIC JAUNDICE

This occurs in a variety of conditions: (1) As a result of poisons, such as phosphorus, arsenic or snake-venom. Experimentally it is readily produced by toluylendiamin. (2) Various specific infections, malaria, pyemia, relapsing, scarlet, typhoid, typhus, or yellow fever. (3) Various obscure conditions, such as acute yellow atrophy, Weil's disease, icterus gravis, etc.

Apart from a slight tinting of the skin and conjunctiva, these cases usually show no symptoms directly referable to the jaundice. The urine shows little or no bile, while the feces have their normal color. In the severer forms the color may be more marked and in some cases there are high fever, delirium, coma or convulsions, black vomit and hemorrhages into the skin.

ACUTE YELLOW ATROPHY OF THE LIVER

Definition.—A rare, acute disease, characterized by widespread necrosis of the liver cells and by jaundice with severe toxic symptoms.

Etiology.—1. It occurs without discoverable cause.

2. It occurs secondarily to pregnancy or to various acute infections,

such as syphilis, pyogenic infections, typhoid or malarial fever, or diphtheria. About one-half the cases occur in women during pregnancy. It occasionally occurs in childhood.

Chloroform and phosphorus-poisoning present similar necrosis of liver cells. The designation, acute yellow atrophy, has been used to cover a group of disorders whose chief point of agreement lies in the acute necrosis of the liver.

Morbid Anatomy.—The liver is one-half to one-third normal size, flaccid, the capsule wrinkled. The color is changed to a greenish-yellow or dirty gray-brown. Subserous hemorrhages may be present. The liver cuts with resistance, the section shows the same color as the surface; hemorrhages may be present. Microscopically there is a general necrosis of the liver cells, with hemorrhages, and round-celled infiltration of the interstitial tissue. The section may hardly be recognizable as liver. The changes in the other organs are not constant. The tissues are usually jaundiced and sometimes show hemorrhages.

Pathogenesis.—The process is now regarded as an autolysis of the liver brought about by the destruction of the liver cells by some unknown poisons, the liver cells being then digested by proteolytic ferments which they normally contain.

Symptoms.—(1) An initial stage, lasting from a few days to a week, of malaise, poor appetite, coated tongue, constipation and occasional vomiting. Slight jaundice is usually present in this stage. (2) Symptoms of intense toxemic jaundice. The jaundice deepens, the vomiting becomes frequent, diarrhea may develop, hemorrhages occur from mucous membranes or into the skin, the patient becomes delirious or comatose, or has frequent convulsions, fails rapidly and dies in coma. A moderate fever may be present in the early stages. At the end the temperature is subnormal and may be so throughout. During this stage the liver dulness diminishes rapidly and may at the end be absent. The urine is reduced in amount and contains, besides albumin and casts, leucin and tyrosin and various organic acids. The amount of urea is reduced, the ammonia relatively increased. The blood shows a leukocytosis and from concentration an increase in red cells.

Diagnosis.—In the first stage this cannot be made. Later the combination of jaundice, severe toxemia, reduction in the size of the liver, and the urinary changes is unmistakable.

Prognosis.—The disease is regularly fatal in from five days to two weeks. A few recoveries have been recorded.

Treatment.—In pregnant women the occurrence of severe toxemia or jaundice should excite suspicion. Careful examination of the urine (nitrogen partition) may then show a grave disturbance of metabolism. Appropriate treatment may avert the development of acute yellow atrophy. These patients must be kept in bed, on a fluid diet, preferably milk, with as much water as possible. The bowels must be kept open. Colon irrigations with warm normal salt solution are useful by increasing the flow of urine and possibly directly increasing elimination. The

treatment of the fully developed condition must be directed to the acid intoxication present. The diet should consist of milk and cereals. The bowels should be kept freely open. Water is given freely. Enteroclysis or hypodermoclysis may be employed. Otherwise the treatment must be symptomatic.

Icterus Neonatorum.—New-born infants may suffer from jaundice. (1) Physiological, so-called. In this form the jaundice develops on the second or third day of life and persists for one or two weeks. Thirty to sixty per cent. of new-born children have it. There are no symptoms other than the pigmentation and no treatment is required. The jaundice is undoubtedly due to bile, as bile-salts may be found in the urine, but the explanation of its occurrence has not yet been given. (2) Persistent jaundice, due either to (a) obliteration of the bile-ducts, (b) syphilitic hepatitis, (c) sepsis, especially that caused by umbilical infection. In the latter form the infants have high fever, rapid pulse, and other evidences of sepsis. Jaundice from these causes persists, the infant gradually loses strength and regularly succumbs. Treatment is of no avail.

DISTURBANCES OF THE HEPATIC CIRCULATION

1. Anemia of the liver is seen post-mortem in fatty or amyloid organs, or after hemorrhage. It produces no clinical symptoms.

2. Hyperemia is common to many diseases:

a. Acute Congestion.—An active hyperemia of the liver regularly follows a full meal, especially if accompanied by alcohol. Acute congestion seems also to be produced by the toxins of certain infections, such as malaria, yellow, typhus and typhoid fevers, erysipelas. The liver dulness may be slightly increased and the edge may be palpable, but there are no definite symptoms.

Treatment.—A calomel or saline purge may be given. Otherwise the underlying condition is to be treated.

b. Passive Congestion. Chronic Congestion. Nutmeg Liver.—This results from obstruction to the return of blood from the liver due to: (1) Failing compensation of the heart, resulting from disease of the valve or muscle, especially when the right side is involved. (2) Obstruction of the pulmonary circulation, such as occurs in emphysema, interstitial pneumonia, large pleuritic effusions or intrathoracic tumors.

Morbid Anatomy.—The liver is enlarged, firm, dark, and bleeds freely on section. The cut surface shows the characteristic nutmeg appearance, from dilatation of the central veins of the lobules, pigmentation of the adjacent cells, and fatty infiltration of those on the periphery. In long-standing cases there may be more or less cirrhosis, both between and within the lobules.

Symptoms.—(1) Fulness or pain in the right hypochondrium. (2) Gastro-intestinal catarrh and hematemesis may result from accompanying congestion of stomach and intestines. (3) Slight jaundice may develop with bile in the urine and clay-colored stools.

PHYSICAL SIGNS.—The liver is enlarged and may be palpable some distance below the costal margin. It may be tender, and in some cases pulsates. The pulsation so caused must be carefully distinguished from that communicated from the heart or aorta.

Treatment.—(1) Free purgative salts may relieve the hepatic congestion. (2) Blood has been aspirated directly from the liver, but this practice is rarely followed. (3) Hematemesis or spontaneous bleeding from piles sometimes accomplishes the desired result.

DISEASES OF THE BLOOD-VESSELS OF THE LIVER

HEMORRHAGE

Bleeding into the substance of the liver may result from (1) trauma, (2) acute yellow atrophy or phosphorus-poisoning, or (3) such diseases as purpura, scurvy, leukemia, malaria, abscess or cancer. The hemorrhages may be single or multiple. In rupture the blood may be poured into the peritoneal cavity and syncope or death result.

EMBOLISM OR THROMBOSIS

(1) Emboli originating in any part of the portal territory may lodge in the liver. Infarction is rarely produced owing to the free anastomoses of the hepatic artery. If the emboli are septic, a suppurative pylephlebitis may result. (2) Thrombosis may occur in (1) cirrhosis, (2) cancer, (3) sclerosis with roughening of the vessel wall, as in Banti's disease, (4) local inflammation from trauma, abscess, or foreign bodies.

Morbid Anatomy.—The condition is that of a more or less complete portal obstruction. The collateral circulation may be well developed. The portal vein may be reduced to a fibrous cord.

Symptoms.—The obstruction, if sufficient, produces the same symptoms as cirrhosis.

Treatment.—The treatment is also that of cirrhosis.

SUPPURATIVE PYLEPHLEBITIS

Etiology.—Suppurative inflammation of the portal vein results from the lodgment in the vein of septic emboli derived from any part of the portal territory, as from gastric or intestinal ulcers, from appendical or pelvic suppurations. Infection of the umbilicus in the new-born may have this result.

Symptoms.—(1) In some cases multiple abscesses in the liver are produced. (See Abscess of the Liver.) (2) In other cases the symptoms are those of pyemia, with an enlarged, tender, and painful liver. The disease lasts for from one to four weeks and is invariably fatal.

Affections of the hepatic artery and vein, such as dilatation, aneurism, embolism, or thrombosis, are met with post-mortem, but are not recognizable during life.

CIRRHOSES OF THE LIVER

PORTAL CIRRHOSIS (LAENNEC'S)

(Chronic Hepatitis. Hobnail Liver. Alcoholic Cirrhosis. Atrophic Cirrhosis)

Definition.—A chronic interstitial inflammation of the liver, resulting in a great increase of the connective tissue, more or less atrophy of the parenchyma, with resulting obstruction to the portal circulation.

Etiology.—This affection is most often seen in men over 40 years, but occurs in women of like age, and is not unknown in childhood. The causes are various: (1) Alcohol is the common one, especially the persistent use of distilled liquors, but wines or beer may act similarly. (2) Acute infectious diseases, such as scarlet fever, typhoid fever, and the like. (3) Tuberculosis and syphilis are occasionally complicated by cirrhosis of the liver. (4) Obstruction of the bile-ducts (chronic jaundice). (5) Toxins produced in the intestine in certain forms of dyspepsia, such as lactic, acetic, or butyric acids, and the like. Such toxins are assumed to account for the cirrhosis of the liver found in splenic anemia and Banti's disease in association with enlargement of the spleen. At least nine-tenths of all cases of cirrhosis of this type are caused by alcohol, which acts either (1) as a direct poison upon the liver cells, causing their degeneration with secondary fibrosis, or (2) by causing gastric and intestinal catarrh which result in the production of poisons having a like effect.

Morbid Anatomy.—There are two typical forms, in both of which the essential feature is a marked increase in the interlobular connective tissue. (1) ATROPHIC CIRRHOSIS: The liver varies greatly in size, sometimes being reduced to one-half the normal, in other cases it is larger than normal. The weight may be similarly reduced, but in the majority of cases exceeds the normal. The surface is rendered rough by contraction of the fibrous tissue, between the bands of which the lobules form prominences, giving the hobnail appearance. The consistency is increased so that the organ feels hard and cuts with resistance. The section shows plainly the thickened strands of grayish connective tissue, between which lie the contracted lobules of liver cells. The color of the liver, both surface and section, is usually a tawny yellow (whence the name). Microscopically the thickened bands of connective tissue are prominent. Within them lie the branches of the portal vein, compressed, thrombosed, or even obliterated. The connective tissue is often densely infiltrated with small round cells. The biliary canaliculi frequently appear greatly increased in numbers. In many cases the growth of new connective tissue is not only interlobular, but intralobular as well. The changes in the liver cells vary greatly, many of them showing degeneration, others hyperplasia and regenerative processes. The reduction in the size of the viscus is doubtless due to the disappearance of many cells. (2) FATTY CIRRHOSIS: In this form the organ is enlarged,

its weight increased, even to ten pounds or more, smooth of surface, more yellow in color. It cuts with less resistance. On section the increased connective-tissue strands appear as in the atrophic form. On microscopic section a widespread fatty infiltration and degeneration of the liver cells is found. In other features it corresponds with the atrophic form.

ASSOCIATED LESIONS.—The peritoneum is thickened and opaque. The spleen is regularly enlarged and hard, showing the changes of chronic congestion. The stomach and intestines are congested throughout, and especially near the esophagus and in the lower rectum greatly dilated veins are found. The kidneys often present the changes of chronic nephritis. Frequently a general arteriosclerosis and corresponding changes in the heart are present. These modifications of the heart, arteries, and kidney are dependent upon the underlying cause of the cirrhosis (alcohol) rather than upon the cirrhosis itself.

COMPENSATORY CIRCULATION.—The portal circulation being obstructed by the cirrhotic process in the liver, the blood seeks new channels of return. These are regularly found in the enlargement of normal anastomoses or possibly by the formation of new vessels. (1) Anastomoses of the gastric and esophageal veins. (2) The veins of Retzius which unite the terminal branches of the intestinal and mesenteric veins with the inferior cava. (3) Anastomoses between the inferior mesenteric and hemorrhoidal veins, branches of the internal iliac. (4) Numerous veins which lie in the suspensory and falciform ligaments and connect the portal system with the mammary and epigastric veins, the accessory portal system of Sappey. Occasionally a large vein, the para-umbilical vein of Sappey, passes along the falciform ligament to unite at the umbilicus with branches of the epigastric veins. Enlargement of these veins about the umbilicus, in rare cases, produces the so-called caput medusæ. (See Fig. 22.)

Symptoms.—These are for the most part due to the portal obstruction. The chronic congestion of the stomach, intestines, spleen, and peritoneum induces appropriate changes of function in each. There is a chronic catarrh of the stomach and intestines which expresses itself as a dyspepsia, sometimes with vomiting and constipation or rarely diarrhea. Such symptoms in an habitual user of alcohol, if accompanied by enlargement of the liver, should always suggest the development of cirrhosis. We cannot, however, venture the diagnosis until more conclusive symptoms of portal obstruction appear. (1) Hematemesis occurs at some time in a large proportion of these patients. It may be the first symptom of the condition. Hemorrhage is caused by rupture of some of the dilated veins of the stomach, especially those about the cardiac orifice, and may be profuse. The bleeding may be repeated. (2) Ascites: Sooner or later in all cases giving symptoms free fluid accumulates in the abdomen. The enlargement of the abdomen is frequently the first complaint. Once begun the effusion usually accumulates rapidly, and if removed returns within a few weeks. Large

accumulations give rise to great distress from weight and pressure upon the adjacent viscera, especially the heart and lungs. (3) Hemorrhoids are frequently caused by dilatation of the anal veins, and free bleeding may occur from them. Hemorrhage either from the stomach or rectum may temporarily relieve the patient. (4) The facies regularly assumes a characteristic appearance, the skin and conjunctiva pale, with a slight icteric tint in the latter, the lids relaxed and puffy, the nose enlarged, red, and covered with dilated veins. Excepting the touch of jaundice these features belong to any chronic alcoholic patient. The jaundice is explained by slight obstruction to the flow of bile through the inter-lobular bile-ducts. (5) Edema of the lower extremities may appear early, either from an associated nephritis or from the pressure of a large quantity of ascitic fluid upon the internal iliac veins and the vena cava. (6) Physical examination shows emaciation with an en-larged abdomen giving characteristic dulness and fluctuation. (See Ascites.) After the removal of the fluid the liver can be felt, its edge hard, its surface rough and granular (hobnail). The spleen also may be palpable. The veins of the abdomen are dilated, but the classic caput medusæ is rarely seen. Hemorrhoids can usually be found. Edema of the feet and legs and the characteristic facies may be present, but are not essential. (7) The urine is scanty, of high specific gravity, and usually contains a trace of albumin and casts. The urea is diminished on account of the lessened activity of the liver. (8) Fever is usually absent, but a moderate temperature, 100°–101° F., may occur at times. (9) Cholemia: Not infrequently the end is marked by increasing mental dulness, stupor, coma, or convulsions, with or without an increase in jaundice, and with a rising temperature. Whether these symptoms are caused by final failure of the function of the liver or by uremia due to nephritis is not at present known. It is evident that just in proportion as the compensatory circulation is established in cirrhosis, the food escapes the detoxicating influence regularly exerted upon it by the liver.

Course.—Usually the gastric symptoms are the earliest, but any of the above symptoms may be the first. If the cirrhosis be marked, ascites regularly appears within a few weeks or months. Once ascites begins it regularly recurs, after steadily shortening intervals, until the patient succumbs to exhaustion or the so-called cholemia. Some few recover, the compensatory circulation developing sufficiently to relieve the portal engorgement. The duration of life after the onset of ascites is usually not more than 18 months to two years.

Diagnosis.—This cannot be made with certainty until the appearance of ascites. The other causes of ascites must then be considered, especially carcinoma of the liver and tubercular peritonitis. Cancer of the liver is usually secondary, gives rise to greater enlargement of the liver, with irregular tumors. Pain is complained of. Cachexia is marked, and secondary deposits may occur elsewhere. Tubercular peritonitis is regu-larly associated with fever, the ascitic fluid is encysted, not free, there
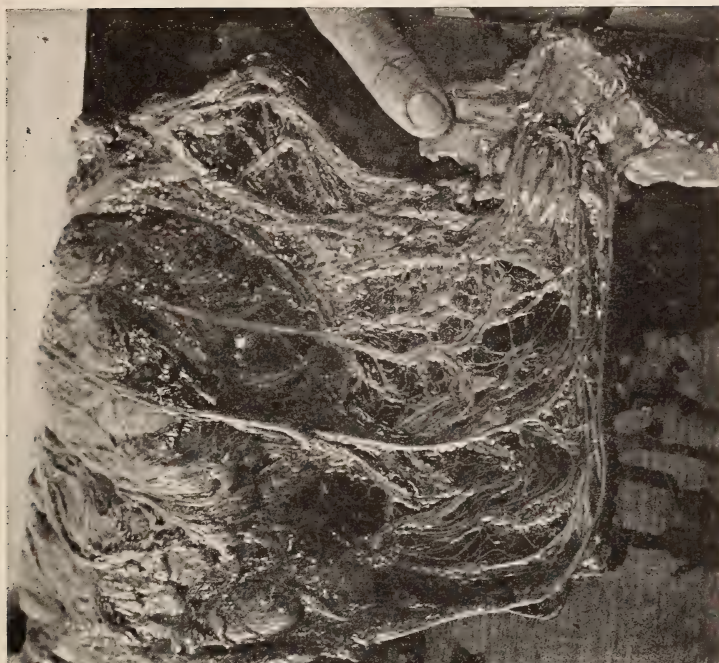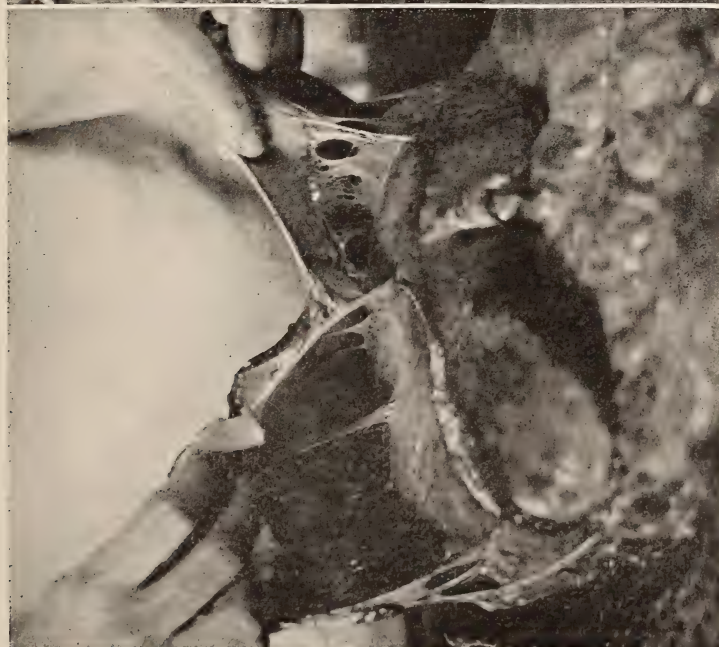
FIG. 22.—The process of natural cure in cirrhosis of the liver. *A*, adhesions between the surface of the liver and the diaphragm in which many fine vessels run; *B*, a leash of vessels in the omentum adherent to a part of the abdominal wall (the tissue held under the fingers). From the collection of Dr. A. R. Lamb.

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are masses or thickenings to be made out in the peritoneum, and the other signs of portal engorgement are lacking. Tuberculosis may be present elsewhere. Finally the examination of the ascitic fluid of cirrhosis shows a specific gravity below 1015, from 1 to 3 per cent. of albumin, and microscopically chiefly endothelial cells with a few leukocytes or blood cells. (See Tubercular Peritonitis.)

Prognosis.—Cirrhosis of the liver is usually fatal in from two to three years from the onset of symptoms, or from one to two years after the development of serious symptoms, such as ascites or hematemesis.

Treatment.—All irritants must be stopped, especially alcohol. With severe gastric symptoms a milk diet may be required. Later the diet should be solid rather than fluid and of easily digestible foods. Nitrogenous foods should be restricted. Constipation must be relieved, preferably by hydragogue cathartics such as the heavy mineral waters or salts, compound jalap powder, or elaterine. Ascites, if slight, may be met by such catharsis and restriction of fluids. Sweating may be employed in strong patients. Tapping is regularly required and must be repeated according to the needs of the patient. A trocar and canula are used. The patient is seated. Strict asepsis must be practiced. Care must be taken to see that the bladder is empty. Then the trocar is introduced in the mid-line midway between the umbilicus and symphysis. As much as 20 quarts may be withdrawn. To avoid any possible danger from the sudden release of pressure, a many-tailed bandage should be applied to the abdomen from above downward, and pressure maintained as the fluid is withdrawn. Talma's operation for drainage and the furtherance of anastomoses has been successful in a number of cases and may be tried in vigorous patients. Otherwise treatment is purely symptomatic.

BILIARY CIRRHOSIS

(Hanot's Cirrhosis)

Definition.—A chronic, probably infectious, disorder of the liver, marked by cirrhosis, enlargement of the spleen and chronic jaundice, without symptoms of portal obstruction.

Etiology.—The disease is frequent in India and rare in the United States. Alcohol probably bears no relation to it. Children of both sexes are frequently affected, while most of the adult cases occur in men. French writers who have devoted most study to the disease believe it infectious, but the active agent is unknown.

Morbid Anatomy.—The liver is very large, weighing from 4.5 to 9 lbs. (2000 to 4000 gms.), its surface smooth and dark olive green in color. The section is firm and of the same color. The connective tissue may be seen in bands outlining the lobes or extending into them.

Microscopically the connective tissue is seen, and the invasion of the lobule is usually marked. In the bands of fibrous tissue are many bile canaliculi showing catarrhal inflammation of their walls. The

number of canaliculi is apparently much above normal. The liver cells show little or no degeneration. The invasion of the lobule by the fibrous tissue and the many fine bile-ducts showing inflammatory changes distinguish the affection from portal cirrhosis.

The spleen is greatly enlarged, weighing 1.3 to 2.6 lbs. (600 to 1200 gms.), and shows fibrosis. All the organs are bile-stained.

Symptoms.—The disease begins in childhood with digestive disturbances or without definite symptoms. (1) Gradually the patient becomes jaundiced and remains so, with all the symptoms of obstructive jaundice, except that the feces are dark and contain bile-pigments. (2) Attacks of pain in the right hypochondrium with fever, leukocytosis, and increase of the jaundice occur from time to time, and continue for days or weeks. (3) The spleen becomes enlarged and firm. Physical examination shows deep jaundice, a large, smooth, sharp-edged liver and spleen, sometimes an enlargement of the tips of the fingers. The blood presents the picture of a secondary anemia with some leukocytosis. The urine shows the presence of bile.

Course.—The disease is slowly progressive, usually lasting from 4 to 5 or 10 years. The patients are likely to die with the symptoms of grave jaundice, high fever, delirium, coma, or convulsions, and hemorrhages.

Diagnosis.—From portal cirrhosis the disease is distinguished by its occurrence in the young and non-alcoholic, the marked jaundice, absence of ascites and other signs of portal obstruction, and chronic course. The attacks of fever with deepening jaundice and leukocytosis are important. Some cases can with difficulty be distinguished from Banti's disease.

Treatment.—The use of antiseptics eliminated in the bile, such as urotropin or the salicylates, is advised. Otherwise the treatment must be symptomatic.

ABSCESS OF THE LIVER

Etiology.—1. The solitary or tropical abscess. (1) Idiopathic cases are not infrequent in this country. (2) Dysentery is the common antecedent. Amebæ, or staphylococci or streptococci may be found in the abscess.

2. Embolic abscesses are common. These arise (1) from infective processes in the territory of the portal vein, such as appendicitis, typhoid fever, dysentery, etc.; (2) from systemic infections, such as ulcerative endocarditis, or pyemia.

3. Trauma either to the body or head may cause a liver abscess.

4. Suppurative cholangitis from any cause may cause multiple abscesses.

5. Foreign bodies, such as needles or fish-bones, or parasites, such as round worms or echinococci, sometimes cause abscesses of the liver.

Morbid Anatomy.—1. The SOLITARY or TROPICAL ABSCESS is usually a single large abscess, either on the surface or in the substance of the right lobe. If the abscess be on or near the surface there is more or less perihepatitis. The abscess cavity is large and filled with pus mixed with blood and broken-down liver tissue, giving it a reddish-brown tinge like anchovy sauce or chocolate. The walls of the abscess are ragged and necrotic. Amebæ may be found in the pus. Cultures may be sterile, or may yield bacillus coli, staphylococci or streptococci or other organisms. These abscesses may rupture into any of the adjacent viscera, the lung, pleura or peritoneum.

In the single traumatic or idiopathic abscesses observed in New York the ameba is not found, but the common pyogenic organisms are obtained in cultures. MULTIPLE ABSCESSES: These are usually small and scattered throughout the liver, which is much enlarged. The surface is smooth. These multiple abscesses may center about the branches of the portal vein, the gall-ducts, the hepatic vein or artery. When the gall-ducts are involved the contents of the abscess are bile-stained, and gall-stones may be found. Suppurating echinococcus cysts are recognized by their contents.

Symptoms.—1. THE SOLITARY ABSCESS.—There are regularly local and constitutional symptoms. (1) Local: (a) Pain, usually dull and aching, is complained of in the liver region. It may be referred to the back and right shoulder. Frequently the patient complains of a dragging sensation in the right side, when he lies upon the left. (b) The liver enlarges and becomes palpable. The increase in size is usually upward and to the right, so that the liver dulness rises abnormally high behind, even to the angle of the scapula. (c) The liver edge is tender to pressure. (d) In rare instances the abscess is so placed superficially that fluctuation can be detected or it may even rupture through the skin. (e) Other local signs may be produced by pressure upon adjacent organs, such as the lungs, whose embarrassment may give rise to dyspnea and cough. (2) Constitutional: These are those of sepsis, a fever of variable type, usually remittent, with more or less rapidity of the pulse, prostration and an increasing anemia. Loss of appetite, nausea or vomiting occurs at times, and there may be either constipation or diarrhea. Weight is ordinarily lost rapidly. The severity of both local and general symptoms varies greatly so that these abscesses are sometimes classified as acute or chronic. The duration varies from one to two months to years. Perforation of the abscess into the lung occurs frequently, and after such relief recovery may occur. The pus discharged has a characteristic appearance and the amebæ may be found in it. Rupture into other viscera, the stomach, intestine or pericardium, may occur with corresponding symptoms.

II. PYEMIC ABSCESSES.—The liver is enlarged, a little tender, and the conjunctivæ present a slight icteric tint, but unless these symptoms are marked the condition is overlooked, by reason of the severity of the constitutional symptoms, which are those of pyemia.

Prognosis.—This is always grave. The mortality is 50 per cent. Many patients succumb even after the abscess has been opened and treated with the greatest surgical skill.

Diagnosis.—From malaria the septic fever ought easily to be distinguished by the examination of the blood and the use of quinine.

From empyema or abscess of the lung, or subphrenic abscess, the diagnosis is very difficult, if there is no history of dysentery or other cause to direct the attention to the liver. Here the line of dulness may suggest the presence of an abscess in the liver rather than fluid in the pleura. The liver may be palpable and very tender, and finally the pus obtained on aspiration may have the characteristics of liver pus and may even contain amebæ.

From intermittent fever associated with gall-stones (intermittent hepatic fever of Charcot) the diagnosis may be made by (1) the long history of the latter, lasting years; (2) the fever, sweating, etc., occur in paroxysms separated by periods of complete freedom from fever; (3) the jaundice deepens after the paroxysms; (4) the general nutrition is maintained.

THE BLOOD.—Leukocytosis may be absent in amebic cases; in septic cases it is usually high, and the differential count gives an increased percentage of the polymorphonuclear cells.

Aspiration of the liver may be employed. The needle may be inserted either in the axilla or behind in the middle of the dull area. A large needle must be used and introduced deeply. An anesthetic may be necessary.

Treatment.—The large solitary abscess must be opened and drained. The small multiple abscesses cannot be treated. If an abscess is discharging through the lung, operation should be delayed, as many patients have recovered under these conditions.

NEW GROWTHS OF THE LIVER

The new growths of clinical importance are carcinoma and sarcoma. Angiomata, adenomata, and other forms of tumor are seen rarely even post-mortem and are not clinically recognizable. Echinococcus cysts, one form of tumor of the liver, are described elsewhere.

Cancer of the Liver.—This is by all means the most frequent form of tumor. Cancer of the liver is primary in less than 5 per cent. of cases. The original focus is most frequently in the stomach, but may be in the gall-bladder, pancreas, rectum or other organs. Cancer develops in persons over 40 years of age, either men or women, and from causes at present unknown.

Morbid Anatomy.—Three forms of primary cancer of the liver are recognized: (1) Nodular, 65 per cent. of all. The cancer occurs in the form of grayish-white, opaque nodules of varying size, scattered throughout the organ. Frequently the nodules are umbilicated. (2) Massive, 23 per cent. of all. In this form there is one large tumor replacing to

a great extent a whole lobe. (3) Infiltrating, 12 per cent. of all. In these cases the appearance is that of a diffuse cirrhosis (portal cirrhosis) of the organ, but on microscopic examination the connective tissue is found to be everywhere infiltrated with cancer cells. Secondary cancer of the liver regularly takes the nodular form described above.

Symptoms.—These may at the outset be due to the primary growth, although in many cases such symptoms are lacking. Regularly a period of failing health, marked by languor, anemia, and possibly emaciation, precedes the development of symptoms which make the diagnosis possible. (1) Tumor of the liver: The organ increases greatly in size, the edge and palpable surface are irregular (nodular). The enlargement is progressive and the liver is tender to pressure. In the massive type a definite tumor may be outlined. In the infiltrating type the liver feels exactly like the cirrhotic. (2) Jaundice regularly develops either from pressure upon the biliary passages outside the liver or from involvement of large numbers of them within the organ. (3) Ascites also develops, due either to pressure upon the portal vein or to involvement of the peritoneum, or in the terminal stages to the cachexia. In addition to these signs we have the symptoms characteristic of cancer in any location. Pain is usually present and is referred to the enlarged liver. Emaciation becomes marked and the cachexia extreme. Fever is absent or slight till the terminal stages: it may then be high. Edema of the feet and legs occurs toward the end, produced either by pressure upon the vena cava or by the anemia and cardiac weakness. In the infiltrating type the symptoms are those of portal cirrhosis, but with more rapid anemia and emaciation. The liver is enlarged, its surface rough, exactly as in cirrhosis. The diagnosis is rarely made during life.

The blood regularly shows an extreme secondary anemia. The red cells may number 2,500,000 or less. The leukocytes may be normal in number or increased, even to 25,000.

DIAGNOSIS.—The constitutional symptoms with the local signs of cancer, when the latter develop, usually enable the diagnosis to be made. Other forms of enlargement of the liver, such as fatty or amyloid liver, cirrhosis, either portal or biliary, syphilis of the liver, or echinococcus cyst must be excluded by careful study. Having made the diagnosis of cancer of the liver, we should in every case seek to locate the primary growth. For this purpose careful investigation of the abdominal viscera should be made.

PROGNOSIS.—The duration of life is given as from four to seven months.

TREATMENT.—Treatment is purely palliative.

SARCOMA of the liver is exceedingly rare. Primary sarcoma is almost unknown. Secondarily the disease follows primary tumors of the skin, the bones, or the eye. It is to be remembered that primary melano sarcoma of the eye is peculiarly likely to be followed by metastasis in the liver. Clinically the disease is in all respects like cancer.

FATTY LIVER

Excessive deposition of fat in the liver may be due either to fatty infiltration or fatty degeneration of the liver cells. In this relation the distinction is not important.

Etiology.—The important causes of fatty liver are (1) obesity, the liver being one of the storehouses of fat; (2) alcoholism; (3) cachectic states, as in the terminal stages of tuberculosis, the severe anemias, cancer, etc.; (4) certain gastro-intestinal disorders, such as occur in children. More or less deposition of fat occurs in the liver under the influence of many other poisons, especially the metallic poisons, arsenic, phosphorus, and the like, but not in sufficient amount to be of practical importance.

Morbid Anatomy.—The liver is enlarged and may weigh 4 or 5 pounds (4000 grammes), the surface pale and smooth, the section yellow and greasy, the consistency diminished. Microscopically the liver cells, especially in the periphery of the lobules, are infiltrated with fat; in some cases the fat droplets apparently replace entirely the liver cells.

Symptoms.—There are none directly attributable to the condition of the liver. On examination the liver is found enlarged, sometimes several centimeters below the costal margin. It is perfectly smooth, not tender or painful.

Diagnosis.—This is based on the recognition of a painless, smooth enlargement of the liver, following the action of some appropriate cause and producing no symptoms. In childhood enlargement of the liver is regularly of this nature.

Treatment.—Treatment is required only for the underlying condition.

AMYLOID LIVER

Amyloid degeneration of the liver is regularly part of such degeneration, involving other viscera, especially the spleen and kidneys.

Etiology.—It is met with most often in children and young adults (under 30 years) as the result of chronic suppuration such as occurs secondarily in tuberculosis and syphilis, especially in those forms of the disease involving the bones and lungs. It does occur in cachexias of other kinds, such as malignant disease, chronic malaria, etc., but rarely in those cases becomes of practical importance.

Morbid Anatomy.—The liver is enlarged, sometimes enormously, its form preserved, the surface smooth and pale, the consistency increased, so that it is firmer and heavier than normal. The cut section is pale, and peculiarly translucent in appearance. Treated with iodine the section becomes mottled with areas stained deep brown—the amyloid tissue. Microscopically the amyloid material is found particularly in the walls of the smaller arteries, and later in the capillaries and venules.

Symptoms.—The patients are emaciated and anemic as the result of the primary trouble. The liver is greatly enlarged, may reach the level of the umbilicus or beyond it, smooth, firmer than normal, not painful or tender. The spleen is similarly enlarged, and usually there are evidences of like degeneration of the kidney, polyuria, with albumin in large amounts and waxy casts. Jaundice is absent.

Diagnosis.—The sequence of protracted suppuration and painless enlargement of the liver, which is smooth but firmer than normal, is characteristic. From fatty liver it can be easily recognized by the associated changes in the spleen and kidney.

Prognosis.—Amyloid degeneration is always a grave sign. The process is progressive and the end not long delayed.

TREATMENT must be directed entirely to the primary condition.

ANOMALIES IN FORM AND POSITION OF LIVER

Malformations of the liver are either congenital or acquired. Congenital malformation may consist of a disproportion in the size of lobes or in an increased lobulation: clinically it is unimportant. Acquired malformation results from (a) tight lacing. The right lobe is compressed by the lower ribs, and its anterior portion is forced downward and presents as a smooth, flat, pyramidal mass (Riedel's lobe) in the right hypochondrium, its edge sometimes reaching the level of the crest of the ilium. The line of pressure of the ribs may be marked only by a groove or by a dense band of cicatricial tissue which may form the only connection between the so-called Riedel's lobe and the rest of the organ. (b) Deformity of the vertebræ or ribs may change the shape of the liver.

SYMPTOMS are lacking, but the distorted liver forming a Riedel's lobe may easily be mistaken for a tumor of any of the adjacent viscera. Its form, consistency and connection with the liver, taken with the absence of symptoms and the sex of the patient, should distinguish it.

Displacements.—1. Congenital.—Transposition of the liver is a rare condition, nearly always part of a complete transposition (situs transversus) of the heart, liver, spleen and other organs. 2. Acquired: The liver may be displaced (a) upward by ascites, abdominal tumors, or distention of the intestines; (b) downward by pleuritic effusions, emphysema of the lungs, or intrathoracic tumors; (c) by relaxation of the ligaments, as in Glenard's disease, the liver is allowed to fall downward and forward.

SYMPTOMS are usually lacking, except those resulting from pressure upon the adjacent organs. In downward displacements the organ may be mistaken for some form of abdominal tumor. Absence of dulness in the normal site and the ease with which the organ can be replaced by pressure usually make its recognition easy.

DISEASES OF THE BILE-PASSAGES AND GALL-BLADDER

ACUTE CATARRH OF THE BILE-DUCTS

(Catarrhal Jaundice.   Acute Catarrhal Angiocholitis)

Definition.—An acute catarrh of the common bile-duct causing obstructive jaundice.

Etiology.—This affection is most frequently seen in young adults, but may occur at any age. It may be due to (1) Extension of catarrhal inflammation from the stomach and duodenum, caused by errors of diet, exposure to cold and wet, malaria, etc., into the common bile-duct. This is the usual cause. (2) Infectious fevers, such as pneumonia or typhoid fever. (3) Emotional disturbances. (4) Congestion due to disease of the heart, kidneys, or lungs. (5) It may occur as an acute infection in epidemic form.

Morbid Anatomy.—Little is known of the pathology. We assume that it has the usual characters of catarrhal inflammation and that the common bile-duct, especially the terminal portion, is blocked either by swelling or by a plug of inspissated mucus. The liver may be enlarged and the gall-bladder distended.

Symptoms.—The onset may be acute with moderate fever, 101° to 102°, nausea, possibly vomiting, and prostration. Usually it is insidious with vague gastric disturbance, the cause of which is revealed by the development of the jaundice. Anorexia, nausea, or vomiting may prevail for several days or even a week before the jaundice. This presents the symptoms of obstructive jaundice, the icterus, bile-tinged urine, clay-colored stools, slow pulse, etc. Drowsiness and languor are usually pronounced. The liver and spleen may be slightly enlarged. The affection lasts from two to four weeks, clearing up very gradually; in some cases it may be protracted to three months.

Diagnosis.—This is the usual explanation of jaundice in young people. In older persons we must think of the other causes of obstructive jaundice, especially gall-stones and cancer. The short duration of catarrhal jaundice and the absence of pain or other symptoms are significant.

Treatment.—Rest in bed is necessary only when the onset is severe. The diet must be suited to the gastric catarrh. Meats, fats, and sweets should be limited or excluded. The bowels require the use of laxatives, especially salines, such as sodium phosphate. Plain water or alkaline mineral waters may be given freely. Theoretically sodium salicylate or sodium bicarbonate may have a favorable influence by thinning the bile.

CHRONIC CATARRHAL ANGIOCHOLITIS

Etiology.—Chronic catarrhal angiocholitis is an accompaniment of chronic obstruction of the bile-ducts by stricture, tumor, pressure from without, or most frequently gall-stones.

Morbid Anatomy.—Behind the obstruction the bile-ducts are distended, their walls thinned or thickened by chronic inflammation. The contents consist of clear, sterile mucus, if the obstruction is complete; when the obstruction is partial only, the mucus is turbid, bile-stained, and yields on culture the colon bacillus or the ordinary pyogenic cocci. The causative factor, such as gall-stones or tumor, is present.

Symptoms.—When the obstruction is complete the symptoms are those of chronic obstructive jaundice. When incomplete the picture is modified by attacks of chills, fever, and sweating (secondary infection) lasting for days or weeks, with intervening periods of freedom from fever. This so-called hepatic intermittent fever is most often caused by gall-stones. The blood shows a leukocytosis.

Treatment.—This must be surgical, the removal of the obstruction.

SUPPURATIVE AND ULCERATIVE ANGIOCHOLITIS

Definition.—A general suppurative inflammation of the bile-ducts, usually accompanied by like conditions in the gall-bladder.

Etiology.—There are regularly two factors. (1) Stagnation of bile, produced by some form of obstruction; (2) Infection with pyogenic organisms. Occasionally only the latter cause is present, the affection following diseases such as pneumonia or typhoid fever. The pneumococcus, the typhoid or colon bacillus, or the ordinary pyogenic organisms may be found in the pus.

Morbid Anatomy.—The ducts and gall-bladder are distended, their walls thickened, the mucous membrane eroded, and they contain pus. Perforation of the distended ducts or gall-bladder may occur, with secondary peritonitis.

Symptoms.—The clinical picture is that of a severe septic infection, with swollen and tender liver, enlarged gall-bladder, jaundice and a high leukocytosis. The distended gall-bladder may be palpable or we may only make out rigidity and tenderness to pressure of the overlying abdominal wall.

Treatment is purely surgical.

ACUTE INFECTIOUS CHOLECYSTITIS

Definition.—An acute inflammation of the gall-bladder produced by bacterial invasion. The affection is similar to that just described, but limited to the gall-bladder.

Etiology.—(1) Most frequently gall-stones are present. (2) Pneumonia or typhoid fever may precede the attack. In its genesis, as well as in its pathology and symptomatology, the disease presents a close analogy to appendicitis.

Morbid Anatomy.—The gall-bladder is distended, its contents consisting of mucus, pus, and blood in varying proportions. There may be a localized peritonitis about the organ, or if its walls are perforated, an

abscess or acute general peritonitis. The pneumococcus, typhoid or colon bacilli, streptococci or staphylococci may be found in the pus.

Symptoms.—Sudden pain in the right side of the abdomen, followed by fever, rapid pulse, nausea and vomiting, mark the onset. The abdomen becomes distended, rigid and tender in the gall-bladder region. Occasionally we may feel the distended gall-bladder. More often the rigidity forbids. There is no jaundice unless the common duct is involved. There is a high leukocytosis. In some cases the picture is much like intestinal obstruction with stoppage of gas and feces.

Diagnosis.—The affection is most often mistaken for appendicitis or intestinal obstruction. From the former it can be differentiated only by its closer relation to typhoid fever or pneumonia, a knowledge of the presence of gall-stones, or ability to feel the distended gall-bladder. Localization of pain, tenderness and rigidity over the gall-bladder region are suggestive, but the appendix may, as we know, lie directly beneath. The acute inflammatory onset and the localization of the signs should exclude intestinal obstruction.

Prognosis.—The milder cases recover within a week under palliative treatment. Severer infections go on to suppuration and may result fatally from sepsis or from secondary peritonitis.

Treatment.—For the milder conditions the application of an ice-bag over the gall-bladder and the relief of abdominal distention by enemata or stupes seem sufficient. If suppuration occurs, the incision and drainage of the gall-bladder or its complete removal may be necessary. The total leukocyte count and the differential are helpful indications of the development of suppuration as in appendicitis.

CHOLELITHIASIS

Etiology.—There are two essential factors in the production of gall-stones: (1) A catarrhal inflammation of the gall-bladder. (2) An obstruction to the free outflow of bile. Gall-bladder inflammation is excited most often by typhoid or colon bacilli, less frequently by the ordinary pyogenic organisms. A previous history of typhoid fever or other acute infectious disease is therefore frequent in cholelithiasis. Stagnation of bile is brought about by a sedentary life, lack of exercise, relaxation of the abdominal muscles, such as follows repeated pregnancies, or in women by tight lacing. While much influence has been attributed to dietetic errors with consequent disturbances of metabolism, there is at present no clear evidence that diet has any direct influence upon the formation of gall-stones. Practically cholelithiasis is met with most frequently in women, and usually in those over 40 years of age. The proportion of women to men affected may be as high as 5 to 1. Seventy-five per cent. of the cases occur in people over 40 years of age and but 1 per cent. in those under 20.

Morbid Anatomy.—Gall-stones may occur singly, but usually are multiple and may number thousands. They vary in size from minute

granules to several inches in diameter; stones weighing 135 grammes are on record. In form they are usually round and smooth, but they may be of various shapes. When multiple they are facetted by friction upon one another. Most of them are quite soft and crumble readily when dried, but some are hard. Large numbers of stones are commonly found in the gall-bladder, the cystic or common duct, or the intestines. Rarely stones are found in the hepatic duct or even in the liver. Sometimes they may be found outside the biliary tract, in adhesions, abscesses, etc.

COMPOSITION OF GALL-STONES.—The stones consist chiefly of cholesterin, bile-pigments, and calcium carbonate. The cholesterin is not derived from the bile, but from the degeneration of the cells lining the gall-bladder. The pigments and calcium salts are derived from the bile itself. The proportion of these several ingredients varies in different stones, but the chief constituent is the cholesterin.

ASSOCIATED LESIONS.—Wherever gall-stones lodge, their presence is associated with some degree of inflammation, catarrhal or suppurative, rarely gangrenous. Thus in the gall-bladder or bile-passages there may be any degree of cholecystitis or cholangitis with their possible results. In some instances the inflammatory process involves the peritoneum and dense adhesions form about the gall-bladder or biliary tract, or in the severer cases there may be localized collections of pus or even a general peritonitis, from rupture of the bile-passages or gall-bladder.

Obstruction may be the chief result of the presence of gall-stones. Either the cystic, or the common duct, or, with very large stones, the intestine may be blocked. If the cystic duct is blocked, the gall-bladder becomes distended, possibly acutely inflamed. In rare instances it atrophies. With the obstruction of the common duct, obstructive jaundice develops. When the stone lodges in the intestine, obstruction of that part results.

Symptoms.—These depend upon two factors: (1) The situation of the stone or stones; (2) the presence or absence of secondary infection. The stones may be in the gall-bladder, the cystic or common duct, the papilla of Vater, or the intestine. The secondary infection may be of the mildest grade, giving evidence of its presence only by the slightest fever and constitutional disturbance at the time of attacks of biliary colic or in the intervals between such attacks, or by symptoms of infection of more marked type, rising at times to the picture of acute sepsis. The situation of the stones does not determine the severity of the infection, but stones in the gall-bladder may be accompanied by violent infections as readily as those in the common duct, or in the papilla of Vater. The results of the combination of the two factors, the mechanical produced by the presence of stones, and the infective, are clinical symptoms of remarkable variety and complexity. In most cases stones are found both in the gall-bladder and in other parts of the biliary tract.

STONES IN THE GALL-BLADDER.—The gall-bladder may be full of stones without producing symptoms, the stones being found at autopsy. In the great majority of cases, however, symptoms result either from

efforts of the gall-bladder to expel the stones, resulting in the classical gall-stone or biliary colic, or secondary infections and inflammation of the gall-bladder.

BILIARY COLIC.—The onset is sudden, the pain is sharp and severe, referred usually to the right hypochondrium, sometimes to the epigastrium, frequently radiating to the back under the right scapula or into the right shoulder, lasting from one or two hours to twelve hours, and followed in many cases after a few hours or a day or two by jaundice. Vomiting regularly occurs at the onset and may be repeated. Relief comes either from the stone dropping back into the gall-bladder or from its passage through the ducts into the intestine. In the first case there should be no jaundice, in the second jaundice is excited by the mechanical obstruction caused at first by the stone itself, later by swelling of the ducts due to traumatism and possibly secondary inflammation. During the attacks there is usually fever, 101° to 103° F., the pulse is rapid, and the patient is prostrated. Fever may be altogether absent. With the subsidence of the pain, recovery is rapid, unless the attack ushers in some of the more remote consequences. These attacks of pain are repeated at intervals of weeks, months or years, depending upon the number of stones present and the efforts of the gall-bladder to eject them. A single attack is a possibility. Repeated attacks regularly impair the nutrition and general health so much as to demand surgical intervention to remove the stones.

With stones in the gall-bladder we may have acute or chronic cholecystitis, with the symptoms already described. The chronic cases are peculiarly likely to be mistaken for dyspepsia or gastritis. The absence of jaundice makes such errors easy. In certain cases even after the escape of the gall-stones the chronic inflammation of the gall-bladder, especially when associated with dense adhesions to the surrounding viscera and abdominal wall, may continue to give symptoms.

GALL-STONES IN THE CYSTIC DUCT.—If the obstruction is complete, the gall-bladder first becomes distended, the bile in it is replaced by clear mucus and ultimately it atrophies and may be reduced to a fibrous mass. If the obstruction is not complete, infective processes develop, and the patient suffers from acute or chronic cholecystitis.

GALL-STONES IN THE COMMON DUCT.—The results depend in part upon the completeness of the obstruction. If complete, the symptoms are those of obstructive jaundice with or without inflammation of the gall-ducts and bladder. (See Cholangitis and Cholecystitis.) Not infrequently in these cases chronic obstructive jaundice is the only symptom. If the obstruction is incomplete, there may be no jaundice at all, or the stone may act as a ball-valve and produce jaundice with periods of remission and exacerbation. Under these conditions secondary infective phenomena are common and the patient suffers from chronic catarrhal or suppurative inflammation of the ducts or the bladder. The latter is the more common and characteristic. With such suppurative inflammation in the gall-passages we may find the so-called hepatic

intermittent fever of Charcot. By this is meant not intermittent fever as commonly understood, but periods of fever, either continuous, remittent or intermittent in type, followed by periods of normal temperature. Increase in the jaundice regularly accompanies the return of the fever, as the swelling of the ducts caused by the increasing inflammation more completely blocks them. In this connection must also be cited the so-called law of Courvoisier. It is not by any means a law, but an observation to the effect that obstruction of the common duct by stone is not followed by dilatation of the gall-bladder, while such dilatation does occur in other forms of chronic obstructive jaundice, such as are produced by tumors in the head of the pancreas, the pressure of tumors upon the ducts, etc. The observation is of help, but like all other observations of this kind it is subject to exceptions.

GALL-STONES IN THE PAPILLA OF VATER.—For the most part the conditions are the same as with stones in the common duct. Stones in the papilla may, however, block the duct of Wirsung, and thus produce accumulation of the pancreatic juice with dilatation of the ducts and possibly a cyst of the pancreas, if the ducts of Santorini are not so situated as to relieve the stoppage. Secondarily chronic inflammation of the head of the pancreas, or pancreatic lithiasis may be met with. If the obstruction of the pancreatic duct is incomplete, bile may be diverted into the pancreas and acute hemorrhagic pancreatitis set up. It will thus be seen that gall-stones may be the cause of important secondary disease of the pancreas. Mayo found gall-stones present as an accompaniment in 81 per cent. of 168 operations for disease of the pancreas.

Diagnosis.—OF BILIARY COLIC.—The diagnosis of the presence of gall-stones often rests on the recognition of attacks of colic. These are identified by the location of the pain, its sudden onset, radiation to the back and shoulder, brief duration, rapid subsidence, and the subsequent jaundice, if present. Jaundice is, however, often missing. In doubtful cases if the feces for 48 hours after the attack are collected and washed through a sieve, the stones may rarely be recovered. Biliary colic must be distinguished from renal colic, gastralgia, gastric ulcer, and appendicitis. If the appendix chance to be located immediately beneath the gall-bladder, an operation may be required to complete the diagnosis.

The diagnostic features of cholecystitis have already been given. In this connection a history of previous gall-stone attacks is of the greatest value.

Gall-stones in the common duct commonly produce jaundice, and must then be differentiated from the various other causes of obstructive jaundice. If jaundice be not present the diagnosis is often extremely difficult. The frequency with which repeated attacks of indigestion in both men and women over 40, but especially the latter, are found to be due to gall-stones must be remembered. Chronic inflammatory lesions of the neighboring viscera, especially the liver, kidney, pancreas, or peritoneum, must be considered. Oftentimes operation alone clears up the diagnosis.

Prognosis.—Gall-stone colic alone is rarely fatal. One attack may be the last, but usually the attacks are repeated. Danger to life arises from the complications, obstructive jaundice, cholecystitis and cholangitis. Where jaundice accompanies the cholecystitis or cholangitis the prognosis, in case of operation, is always grave by reason of the tendency to hemorrhage. Otherwise the danger is in proportion to the severity of the inflammation, *i.e.*, it is greater if pus be present.

The Blood.—The leukocyte count in gall-stone colic will show slight increase or remain normal. If cholecystitis or cholangitis develop, the leukocytes are increased usually in proportion to the severity of the process. If pus be present, the percentage of polymorphonuclears may be increased to 85 per cent. or 90 per cent.

Treatment.—For gall-stone colic the surest relief is had from the hypodermic injection of morphine sulphate, one quarter to one-third grain. Chloroform may be given sufficiently to dull the pain. Hot fomentations to the gall-bladder region are grateful. After the attack we must face the problem of preventing further attacks or the formation of more stones. The patient must be given an easily digestible diet. As already explained, the cholesterin does not come from the food, nor has the composition of the food much influence upon the biliary secretion. The diet is therefore to contain proteids, fats, and carbohydrates in reasonable proportions and in forms tending to their ready digestion. Alkaline waters are given freely. Exercise is required, especially such as bring the abdominal muscles into play. The bowels must be kept open, by laxatives if necessary. Sodium phosphate or sulphate may be given in one to two dram doses in a glass of water each morning. For repeated attacks of gall-stone colic or for the severer complications of gall-stones surgical intervention is necessary. The milder forms of these affections may yield to rest in bed, regulation of the diet, relief of constipation, and the local application of an ice-bag.

DISEASES OF THE PANCREAS

I. ACUTE PANCREATITIS

ACUTE HEMORRHAGIC PANCREATITIS

Etiology.—The disease occurs most often in men, especially those over forty. But two definite causes are known: (a) Trauma, such as blows upon the abdomen, and (b) obstruction of the papilla of Vater by gall-stones so that bile is forced into the pancreatic duct. In many cases no satisfactory cause is found. Bacterial invasion, especially by the bacillus coli, can often be demonstrated, but is probably secondary.

Morbid Anatomy.—The pancreas is enlarged, firm, dark-red, and marbled by the mingling of hemorrhagic areas and normal tissue. The interstitial tissue is infiltrated with blood, the organ ruptured in places, and the blood infiltrates the retroperitoneal tissue and forms a more or less extensive hemorrhage, in some cases extending even into the pelvis. The peritoneum may contain bloody serum. The subperitoneal

tissue of the abdominal walls, the mesocolon, the mesentery, and retro-peritoneal fat, show areas of fat necrosis. Microscopically the pancreas shows more or less extensive necrosis, and hemorrhage, and in places an inflammatory exudate of leukocytes and fibrin. If the course of the disease is protracted to a fortnight or longer the pancreas and adjacent tissues may be found gangrenous.

Symptoms.—The disease may attack robust men or occur in those who have suffered attacks of pain due either to gastric disturbances or gall-stone colic. (See pp. 136–137.) The onset is sudden, marked by severe abdominal pain, referred to the epigastrium, nausea and vomiting. The abdomen, especially the epigastric region, becomes distended, exquisitely tender, and tympanitic. Symptoms of collapse develop rapidly. The patient is profoundly prostrated, pale, the respiration rapid and deep (air-hunger), the pulse rapid and feeble. The temperature may be either normal, subnormal, or elevated. The bowels are usually constipated, but may be loose. In most cases death occurs within one or two days in syncope or coma. Glycosuria is rarely found.

Gangrenous pancreatitis is now recognized only as an advanced stage of the acute form. If the patient survives three or four days, the pancreatic tissues may become gangrenous, and an abscess cavity form in which lies more or less of the necrotic organ.

Symptoms.—The violent pains of the onset subside, the patient develops fever, sweating, rapid pulse and other signs of bacterial invasion. The abdomen continues distended and tender and a mass may be made out in the epigastric region. Jaundice may develop, and glycosuria is occasionally found. In two cases the separated pancreas has been discharged per rectum.

The blood shows a leukocytosis, sometimes quite high, 30,000 or over.

Diagnosis is difficult. The symptoms very closely resemble those of acute peritonitis following the perforation of a gastric ulcer, or those of intestinal obstruction. If the hemorrhage be large enough to give the typical signs of bleeding, the diagnosis may be easy.

Treatment.—For the violent pain morphine may be given hypodermatically or chloroform by inhalation. The colon may be emptied by enemata and stimulating injections given. Hypodermoclysis may be helpful in prolonging life. Surgical treatment offers the only hope of meeting the conditions satisfactorily.

SUPPURATIVE PANCREATITIS

Etiology.—Suppuration may follow acute hemorrhagic pancreatitis or may be primary. Infection takes place either through the blood vessels or ducts. In the latter case, suppuration is greatly favored by obstruction of the duct of Wirsung by gall-stones, cyst, or cancer of the pancreas. It is met with especially in men over forty.

Morbid Anatomy.—The abscess may be either in the substance of the pancreas or in the surrounding tissues. The cavity of the lesser peritoneum may be involved in the process.

Symptoms.—The onset may be that of acute hemorrhagic pancreatitis or it may be gradual with a history of pain in the epigastrium and disturbance of digestion. Only when suppuration is well established, after four or five days, is the clinical picture definite. (1) The constitutional picture is that of sepsis with fever, rapid pulse, sweating, anemia, and emaciation. These symptoms may be slight or very marked. (2) The local signs include distention of the epigastrium with pain, tenderness, rigidity, and possibly an ill-defined tumor lying behind the stomach. Only in rare cases can the tumor be made out definitely. Glycosuria or fatty diarrhea is rare.

Course.—The duration may be a few days or several months. Eventually the abscess bursts into one of the adjacent organs or into the peritoneum. General peritonitis may result from such rupture or may be caused by extension without rupture.

The BLOOD shows a leukocytosis, with possibly an increased percentage of polynuclear cells.

The DIAGNOSIS in the acute stages cannot be made from the acute hemorrhagic type. In the more chronic cases the constitutional symptoms of sepsis with the indefinite tumor in the epigastrium may identify the affection. Such symptoms are of special import if developed subsequently to cholelithiasis, cancer or cyst of the pancreas.

TREATMENT is purely surgical.

CHRONIC PANCREATITIS

A chronic interstitial inflammation of the pancreas, rarely recognized during life, found frequently at autopsy, and of interest chiefly by reason of its relation to diabetes and of the possibility of mistaking it for carcinoma.

Etiology.—(1) Obstructions of the pancreatic duct caused by calculi, either pancreatic or biliary, cysts, or cancer of the gland. (2) Arteriosclerosis, especially that produced by alcohol. In this relation it may accompany cirrhosis of the liver.

Morbid Anatomy.—The pancreas is of normal size or contracted, firm, dense on section. The increased fibrous tissue may be plainly seen. Small cysts may be formed in the gland. Microscopically the increased fibrous tissue is found to be either interlobular or interacinar. In the former case the islands of Langerhans are preserved, in the latter they are atrophic, possibly fibrous.

Symptoms.—Gastric and intestinal indigestion are present, sometimes with attacks of pain, fever and vomiting. The stools may show abundance of fat and undigested muscle fibre, and in severe cases of the interacinar type diabetes may develop. The urine may give Cammidge's reaction. Not infrequently at operations for gall-stones the head of the pancreas is found so hard and seemingly enlarged that it is thought to be cancerous, but observation or autopsy proves the condition to have been chronic pancreatitis. The possibility that such glands may be felt as abdominal tumors in thin persons must be admitted.

PANCREATIC CYST

Etiology.—A rare form of abdominal tumor, seen in adult men and women. It may be caused (a) by trauma; (b) by obstruction of the pancreatic duct due to stone, tumor, or pressure from without; (3) by proliferation of the epithelial tissue of the gland.

Morbid Anatomy.—The cyst forms a round or oval tumor in the upper abdomen, presenting either above the stomach, between stomach and colon, or below the colon. The contents may be clear and watery, or viscid and brownish from the presence of mucus and blood. Microscopically the fluid shows epithelial cells, leukocytes, some of them containing fat, crystals of fatty acids, and altered red cells.

The fluid may show the presence of tryptic, diastatic, and emulsifying ferments; most often the diastatic only is found.

Symptoms.—(1) The tumor is the essential symptom. It presents as a smooth, round or oval mass in the epigastrium, but slightly tender or painful, either movable or fixed; fluctuation may be detected, only if the tumor is near the surface. It may be above or below the stomach, possibly even below the colon. The tumor may be to right or left of the median line, or even in the region of the left kidney, depending upon the portion of the pancreas from which it arises. It may be small or large enough to fill the abdomen. (2) Glycosuria or fatty stools from interference with the function of the gland are rare. (3) Pressure symptoms develop, if the tumor be large, abdominal discomfort or pain referred to the epigastrium; disturbances of the digestion, such as loss of appetite, pain after eating, nausea or even vomiting; in case the tumor arise from the head of the pancreas jaundice may be caused, or ascites from pressure on the portal vein. (4) Loss of weight and weakness are generally noted. These cysts may empty spontaneously into the intestine and later refill. Traumatic rupture into the peritoneum may occur.

DIAGNOSIS rests upon the physical signs of the character and location of the tumor. Distention of the stomach and colon aid in the location. The tumor must be differentiated from an enlarged gall-bladder, cysts of the omentum, hydronephrosis of the left kidney, and in case of very large tumors from ovarian cysts.

TREATMENT is purely surgical. The cyst must be opened and drained.

CARCINOMA OF THE PANCREAS

Etiology.—Cancer of the pancreas occurs in middle life, in men more often than in women. It may be primary or secondary to cancer of adjacent organs, especially the stomach.

Morbid Anatomy.—The tumor most frequently occurs in the head of the pancreas, but may occupy any part. The cancer is usually very hard, scirrhous in type, but may be soft or encephaloid, or colloid. The gland is more or less enlarged, especially the head, dense and hard. On

section the increased fibrous tissue is evident and also gray areas representing the cancerous infiltration. Sharply outlined tumors are rare.

Symptoms.—The development of the new growth is marked by indefinite symptoms of digestive disturbance, loss of appetite, distress after eating, eructations, nausea or even vomiting. After these symptoms have existed for some weeks or months the patient develops the general symptoms of cancer, pain, cachexia, and tumor. (1) The pain is referred to the abdomen, radiating into the shoulders or back. It is more or less constant. (2) The cachexia is marked, loss of both weight and strength being rapid. (3) A tumor may not be palpable for some time, in some cases not at all. Long before it is palpable it gives evidence of its presence by obstructing the common bile-ducts and causing jaundice. The jaundice in these cases deepens steadily till it becomes most intense, and so continues. The gall-bladder may be distended. If palpable the tumor presents most often in the epigastrium, rarely in either hypochondrium. It is hard, tender, and fixed or but slightly movable. Remote evidences of involvement of the pancreas may be found in glycosuria or rarely in the occurrence of fatty stools. The temperature is usually normal or below; fever is rarely seen.

Diagnosis.—The age of the patient, the pain and cachexia, increasing jaundice, and the epigastric tumor are most important. Before the appearance of the tumor the diagnosis from gall-stones is difficult. The history of previous colic, variability of the jaundice, absence of enlargement of the gall-bladder (Courvoisier's law), and presence of fever point to gall-stones. Severe pain, rapid emaciation, steadily increasing jaundice, and distention of the gall-bladder belong to cancer of the pancreas. Glycosuria or fatty stools aid the diagnosis in some cases. The development of a tumor usually settles the question. Cancer of the pancreas must be distinguished also from cancer of stomach or gall-bladder. Chronic interstitial pancreatitis is occasionally at operation mistaken for cancer.

TREATMENT must be purely symptomatic.

PANCREATIC CALCULI

These are exceedingly rare. Only seven cases in which the diagnosis has been made during life are on record. Pancreatic calculi are doubtless produced by causes similar to those acting in cholelithiasis, infection and obstruction. They may therefore accompany tumors and other forms of obstruction. The symptoms, if any be present, cannot be distinguished from those of biliary colic, except by the recovery of the stones. These are found to consist almost wholly of calcium salts, the carbonate and phosphate, without bile-salts or pigment. The treatment of the condition is purely symptomatic.

DISEASES OF THE PERITONEUM

ACUTE GENERAL PERITONITIS

Definition.—An acute inflammation of the peritoneum.

Etiology.—This disease may be either primary or secondary. (1) Primary: The possibility of a primary peritonitis is admitted by all writers, but it is so rare as to be practically negligible. Exposure to cold and wet, and rheumatism are said to cause it. (2) Secondary: The peritonitis is here due to infection, pathogenic organisms being admitted either from without, or from the viscera covered by the peritoneum or in close relation thereto, or in rare instances from the blood. (a) Wounds or operations involving the peritoneum are common causes, especially if they at the same time involve any of the hollow viscera, particularly the stomach or intestines. (b) Perforation by ulceration or new growth of the stomach, intestine, appendix or other hollow viscus, discharging its contents into the peritoneum, or rupture of an abscess of the appendix, gall-bladder, liver, spleen, kidney, tubes, or uterus, or any part of the abdominal cavity into the peritoneum. (c) Extension of inflammation without actual rupture in any of the conditions just given. In cancer of the stomach or intestine, in appendicitis, etc., peritonitis may develop from extension alone. In rare instances the inflammation may extend from the pleura or pericardium. Apart from direct wounds, operative or accidental, acute peritonitis most frequently develops in men from appendicitis, in women from infection through the uterus, tubes and ovaries, from gastric ulcer, or from appendicitis. Acute peritonitis may also be found as a terminal complication in Bright's disease, arteriosclerosis, or gout.

BACTERIOLOGY.—The organisms found include a wide variety. The colon bacillus is regularly present when the infection arises from the alimentary tract, either alone or combined with other bacteria. When abscesses or suppurations of other viscera are the source, streptococci or staphylococci may be expected. Many other organisms may be found usually accompanying these common invaders, rarely alone, such as the pneumococcus, bacillus proteus, bacillus pyocyaneus, bacillus tuberculosis, bacillus aërogenes capsulatus, bacillus typhosus, and gonococcus.

Morbid Anatomy.—The peritoneum is congested, its surface dull and coated with an exudation of fibrin. This may be sufficient only to render the surface granular or may be abundant, gluing the intestinal coils and other parts of the peritoneum together and showing as a thick pellicle. Fluid accumulates in the peritoneum, usually in small quantity, but sometimes to the amount of several quarts. It is serous, sero-fibrinous, or purulent; in rare cases hemorrhagic. Especially in infections from the intestinal tract the exudate presents a very foul odor from the presence of putrefactive bacteria.

Symptoms.—The onset varies greatly. In cases of perforation as in typhoid fever there may be an initial fall of temperature to normal or

below, quickly followed by a rise to 103° or 104°. In other cases the onset may be marked by a chill and rapid rise of temperature, or the onset may be insidious with very little fever. Pain in the abdomen is often the first symptom, sudden, sharp, severe and continuous. Tenderness regularly develops with it. Nausea and vomiting follow, and the vomiting is repeated, the vomitus being first gastric contents, then mucus, and finally bile-stained (greenish) material from the intestine. The bowels are constipated, rarely loose. With these symptoms the patient is greatly prostrated, the pulse rapid, small, and hard, the respiration shallow and somewhat rapid, the face showing suffering and anxiety. After the onset there may be a temporary improvement in the symptoms, but this is soon followed by their return in increasing severity. The temperature may be low, not over 101° F., but usually rises steadily to 104°–106° F. The pulse grows more and more rapid, 140 to 150 per minute, harder and more thread-like; the respiration more shallow and hurried. The facies becomes drawn, the eyes sunken and dark-ringed, the color ashen or livid—the so-called Hippocratic facies. The vomiting persists, the abdomen continues tender and painful and gradually becomes more and more distended. Constipation is marked. There may be frequent urination, difficulty in passing the urine or retention. The urine is scanty and contains a trace of albumin, possibly casts, and much indican. The mind is usually clear until death, but in typhoid fever the patient may be so dull that he complains of no pain and never realizes the change in his condition.

PHYSICAL SIGNS.—The patient lies upon his back with the knees drawn up to relax the abdomen. The abdomen is held rigid, the thorax only moving in respiration. At first the abdomen is sunken, later distended. There is a general muscular rigidity with exquisite tenderness. At the onset the location of the pain, rigidity or tenderness is most marked at the site where the process began, e.g., about the stomach or appendix. When the abdomen is distended, it is markedly tympanitic. Late in the disease there may be sufficient fluid in the flanks to give movable dulness. If gas is present in the abdominal cavity, as from rupture of the stomach or intestine, the liver dulness may be notably diminished or absent, a sign often emphasized, but sometimes seen in other conditions as well. If the abdomen is distended the liver dulness and also the situation of the apex of the heart may be displaced upward.

COURSE AND PROGNOSIS.—The disease is regularly progressive and swiftly fatal. A patient with perforation during typhoid fever usually survives not more than 24 or 48 hours. In other conditions the duration is longer and the patient may live a week. Unless relief is secured by operative procedure, death is regularly the outcome. Recovery is, however, possible, especially in gonococcus or pneumococcus infections.

The BLOOD shows, as a rule, leukocytosis which may be quite high, 20,000–30,000, but this sign may be lacking in the severest cases, especially after perforation in typhoid fever.

Diagnosis.—The combination of the constitutional and local signs is

usually·characteristic. Intestinal obstruction or acute pancreatitis may give a clinical picture which is hardly distinguishable. Error in these cases is not important, for operative interference is called for as much as in acute peritonitis. Great difficulty may be met in certain cases of localized peritonitis, appendical or pelvic, in excluding general peritonitis. The localization of the signs and the general condition of the patient are most important. A few hours' observation with expectant treatment usually leads to a correct conclusion. The greatest difficulty is met with in determining the presence of acute peritonitis following perforation in typhoid fever. The mental condition of the patient often prevents complaint of pain and tenderness. We must then rely upon evidence of a sudden change for the worse in the patient's condition and rapid change in the abdominal condition, with the presence of general rigidity, distention, and possibly fluid in the flanks. Obliteration of liver dulness is suggestive but not conclusive. Hysteria is said at times to give most deceptive signs of peritonitis. A normal blood count would here be most suggestive.

Treatment.—Symptomatic treatment may be employed till such time as the diagnosis is established. Persistent vomiting requires the cessation of mouth feeding, or the giving of food and water in teaspoonful quantities at frequent intervals. Peptonized milk is the best nutriment. If these measures fail, washing out the stomach may give relief. The bowels are emptied by enemata, and if the distention is marked a rectal tube is kept constantly in place, while turpentine stupes are applied to the abdomen. Cardiac stimulants are required and those which can be given hypodermatically are to be preferred, such as digitalin, strychnine, caffeine, and camphor. The severe pain may call for the use of morphine hypodermatically, but in the initial stages the administration of morphine is always most objectionable, because it so masks symptoms as to frequently mislead the observer, and delay the recognition of the gravity of the disease. When the diagnosis is settled and the course of action clear, that objection no longer holds. The practice of giving saline purgatives with the purpose of draining the peritoneum through their action has been given up. As soon as the diagnosis is established the condition must be treated surgically.

LOCALIZED PERITONITIS

Various parts of the peritoneum may be involved in a localized peritonitis. Thus the spleen is frequently bound down by adhesions representing a previous peritonitis, or the liver or gall-bladder may be similarly affected. In women the pelvic organs are most often involved. Most frequently these localized processes are incidents of a major process, such as uterine inflammation or salpingitis, cholecystitis, etc., but occasionally the localized peritonitis becomes of considerable importance. Three principal forms may be described, depending upon the localization of the disease—pelvic, appendical, and subphrenic.

PELVIC PERITONITIS is secondary to disease of the uterus or tubes. It may be septic, gonorrheal or tubercular. It is at first localized and usually remains so, but may become general, or may give rise to abscesses which later rupture into the peritoneum and excite a general process. With the subsidence of pelvic peritonitis adhesions and thickening of the peritoneum are left and persist for years, possibly for life.

APPENDICAL PERITONITIS is a similar process localized about the appendix. It is regularly part of the several varieties of appendicitis, and is produced ordinarily by extension of the inflammation through the wall of the appendix. The abscesses so often found about the appendix are produced in this way. These abscesses remain localized or in some cases rupture into the peritoneum. The adhesions so often found about the appendix are evidences of previous local peritonitis.

The symptoms and treatment of pelvic and appendical peritonitis cannot be separated from those of the underlying condition.

SUBPHRENIC PERITONITIS, SUBPHRENIC ABSCESS or SUBPHRENIC PYO-PNEUMOTHORAX.—A rare form of localized peritonitis involving the peritoneum covering the diaphragm and liver, in some cases possibly the lesser peritoneal sac.

ETIOLOGY.—Primary cases are said to follow injury, but are exceedingly rare. The usual causes are: (1) Extension from abscess of the liver, gall-bladder, right kidney, or appendix. (2) Extension from the pleura. (3) Perforation of a gastric or intestinal ulcer. In these cases air or gas as well as pus is found in the abscess.

Morbid Anatomy.—The abscess on the right lies between the upper surface of the liver and the diaphragm, on the left between the diaphragm and the stomach and spleen, possibly in the lesser peritoneal cavity. The contents consist of pus alone, or in cases arising from perforation of the stomach or intestine, pus with air or gas and even food.

Symptoms.—The general symptoms are those of suppuration, fever, either low or high, rapid pulse, sweating, anemia, prostration and emaciation. From upward displacement of the diaphragm or involvement of the pleura dyspnea may be severe. Local pain and tenderness may be marked.

Physical Signs.—(1) Downward displacement of the liver, if the abscess is right-sided. (2) Enlargement of the thorax. (3) Dulness corresponding to the area of the abscess, or, if air be present, the signs of pneumothorax. (4) If the abscess be on the left side, it may form a tumor in the epigastrium.

Diagnosis.—Subphrenic abscess is often mistaken for empyema, or abscess of the liver, or in the gaseous cases for pyopneumothorax. The condition in the milder forms may be easily overlooked. The aspirating needle is of great aid in locating the pus. For differentiation emphasis must be put on (1) previous history of disease of appendix, gall-bladder, stomach or other source of infection below the diaphragm. (2) Examination by X-rays, which may locate the diaphragm above the abscess.

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(3) Pfuhl's sign: If an aspirating needle is in the abscess, the flow of pus is not interrupted by inspiration, as it is in empyema. (4) Furbringer's sign: If an aspiratory needle is introduced into a cavity above the diaphragm no movement occurs in respiration. If the needle passes through the diaphragm the outer end moves up in inspiration and down in expiration.

Prognosis.—Unless recognized and drained these abscesses are always fatal. In some cases death ensues even after the abscess has been drained.

TREATMENT is purely surgical.

CHRONIC PERITONITIS

Chronic peritonitis is of pathological rather than of clinical interest. It is frequently seen in one form or another post-mortem, rarely recognized during life. Several forms are described.

Local Adhesive Peritonitis.—Local inflammations of the peritoneum are common during life, about the uterus and tubes, the appendix, the liver, spleen, or gall-bladder. As a sequel the peritoneum in any of these localities may be found thickened and adherent. Such adhesions may give rise to occasional twinges of pain during life; occasionally they become important by contracting in such a way as to obstruct the bowel, or interfere with the functions of the stomach, or other viscera.

General adhesive peritonitis is frequently found in association with hepatic cirrhosis or chronic congestion. The peritoneum is everywhere thickened, opaque, and adherent, the coils of intestine everywhere matted together, and adjacent viscera bound down to such an extent that the cavity may be obliterated. In other cases a serous effusion of considerable amount may be present. Such chronic peritonitis may be one factor in the recurring ascites of cirrhosis or chronic congestion. Occasionally the contents of the sac become purulent. We may suspect such a condition where an ascites requires frequent tapping, but cannot prove it.

Proliferative peritonitis is also associated with cirrhosis or chronic congestion, and is very much like the preceding except that in this condition in addition to the general thickening and the serous effusion there are masses consisting of thickened omentum or folds of peritoneum and coils of intestine, the mesentery or mesocolon. These masses may form veritable tumors. Under these conditions tubercular peritonitis would be suspected, but the fluid if removed is found not to contain bacilli or be infective on inoculation into guinea-pigs.

Chronic hemorrhagic peritonitis is a very rare form analogous to hemorrhagic pachymeningitis. The peritoneum in various parts, especially in the pelvis, is found covered with delicate layers of new-formed tissue containing many blood-vessels, and a hemorrhagic exudate may be present.

SYMPTOMS.—These are very vague and difficult to interpret. There

are usually indigestion and constipation and possibly some pain. As already indicated, in some cases there may be an abundant ascites, and the conditions under which chronic peritonitis may be suspected have been indicated.

TREATMENT.—This is usually involved in the care of the associated cirrhosis or cardiac lesion. Otherwise it must be purely symptomatic.

NEW GROWTHS OF THE PERITONEUM

CANCER OF THE PERITONEUM

Various new growths of the peritoneum have been described; of these cancer alone is frequent enough to be of clinical importance. Cancer is rarely, if ever, primary in the peritoneum, but may be found secondarily to cancer in the abdominal organs, especially the uterus, stomach, or intestine. It occurs at the usual age and chiefly in women.

The SYMPTOMS include the constitutional cachexia of cancer with abdominal pain, and the local signs of ascites. Fluid withdrawn may be bloody and may show cells with mitoses. After withdrawal of the fluid one or more masses may be felt in the abdomen. The condition is with difficulty distinguished from tuberculosis. Evidence of a primary growth in other parts, especially the uterus or stomach, must be sought.

TTREATMENT is purely palliative.

ASCITES

(Hydroperitoneum)

Definition.—An accumulation of serous fluid in the peritoneal cavity.

Etiology.—Ascites may be the result of local or general conditions. Local: (1) chronic disease of the peritoneum, simple, tubercular, or cancerous; (2) obstruction to the portal circulation, such as occurs in cirrhosis, cancer, or other disease of the liver, in thrombosis of the portal vein, in chronic congestion due to cardiac insufficiency of any kind, or to chronic pulmonary disease, especially emphysema or tuberculosis; (3) the growth of tumors in the abdomen which by pressure on lymphatics or blood-vessels may lead to ascites. General: ascites occurs as part of a general dropsy in heart disease, Bright's disease, and in the final stages of severe anemia or cachexia, such as occurs in leukemia, cancer, and the like. The dropsy of heart disease is sometimes confined to the abdomen.

Symptoms.—The girth of the abdomen gradually increases and the patient complains of weight or oppression. With large effusions the diaphragm is pushed upward, the lungs compressed and the patient suffers from dyspnea. Gastro-intestinal disturbances, loss of appetite and constipation are regularly present, and the urine is scanty. The associated symptoms are those due to the primary affection.

PHYSICAL SIGNS.—Inspection: the abdomen is prominent, smooth,

symmetrical. Usually the veins over it are dilated and sometimes vari-
cose. If much stretched, the skin is shiny and perhaps shows striæ. The
umbilicus may bulge, especially in children. Palpation: the abdomen
is tense or soft, depending upon the quantity of fluid present. A fluid
wave may be obtained by placing the palm of one hand flat on one side
of the abdomen and then lightly tapping the other side with the fingers
of the other hand. To prevent confusion from the transmission of the
impulse through the abdominal wall it is well to have a third person lay
the ulnar border of one hand on the middle line of the abdomen and
press gently downward, while the test is made. Percussion: the lower
parts of the abdomen are dull, while the upper are tympanitic. Thus,
the patient lying upon his back, the flanks are dull, the central parts,
occupied by stomach and intestine, tympanitic. (See Fig. 23.) If the
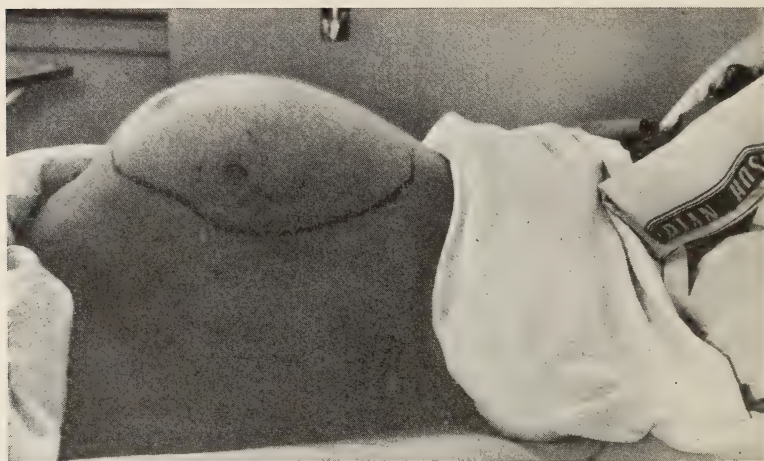
Fig. 23.—An ascitic abdomen: flatness below the line, tympany above. From the collection of Dr.
A. R. Lamb.

patient stands or sits the dulness lies across the lower abdomen. If he
turns on one side, the lower side becomes dull, the upper tympanitic.
This shifting dulness is very characteristic.

Diagnosis.—A distended bladder, large cysts, especially ovarian,
and pregnancy with excessive amniotic fluid may be sources of error.
None of the conditions named give the shifting dulness characteristic
of ascites. The dulness is central, or unilateral, the flanks clear, and
there is no material change with change of position.

Character of Fluid.—This is usually clear serum, alkaline, of a
specific gravity of 1.010 to 1.015, and containing a large amount of
albumin. It may coagulate spontaneously. The fluid may be bloody,
especially in tuberculosis or cancer, or chylous. The latter may arise
from the presence of true chyle (very rarely) or from a fatty degenera-
tion of cells present in the exudate. The study of the cellular content

of the fluid is helpful. In simple ascites the cells are chiefly endothelial, in tuberculosis lymphocytes will predominate, in cancer mitotic nuclei may be found.

Treatment.—The reduction of the fluid in the abdomen may be sought by reducing the intake of water and increasing the output by hydragogue cathartics and diuretics. Large doses of magnesium sulphate, or Epsom salts, or compound jalap powder may be given on rising. The effusion of digitalis or diuretin may be given for diuresis. Frequently these measures fail and tapping by trocar and canula must be resorted to and repeated as necessary. The puncture is regularly made in the mid-line midway between the umbilicus and the pubes. Care must always be taken to see that the bladder is first emptied.

DISEASES OF THE KIDNEYS

ANOMALIES OF FORM AND POSITION

Post-mortem observation has established the possibility of a large number of anomalies of the kidney. Those of clinical importance are: (1) Total absence of one kidney, a not very rare occurrence. The single kidney is usually considerably larger than normal. There may be a patent ureter on the side of the absent kidney. The rule is now established that before undertaking a nephrectomy the surgeon should make sure of the presence of a second organ. (2) Displacement of the kidney. Any part of the abdomen or pelvis may be occupied by
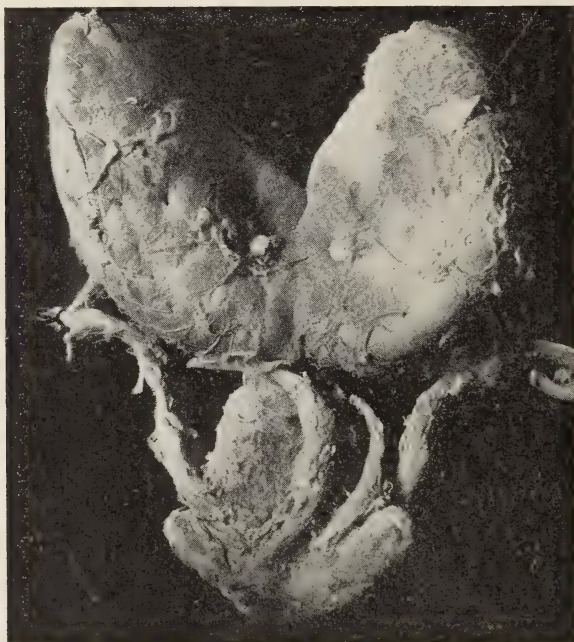
Fig. 24.—Horse-shoe kidney.

the displaced viscus. The kidney may be fixed in the abnormal situation and may thus be mistaken for a tumor. (3) Double ureters may be found on one or both sides, a possibility of importance in cystoscopy. (4) Horse-shoe kidney. Not infrequently the lower poles of the two kidneys are fused, producing a form resembling a horse-shoe. This deformity is of importance from the possibility that the fused organ may be mistaken for a tumor, or may cause difficulty in operations upon the organ. (See Fig. 24.)

MOVABLE KIDNEY

(Floating or Palpable Kidney. Nephroptosis)

Definition.—An abnormal mobility of the kidney. Under ordinary conditions the kidneys are not palpable. If, on deep respiration, the lower pole of the kidney can be appreciated by the palpating fingers, the kidney is said to be palpable. If the fingers can be slipped over the upper pole, the kidney is movable. In some instances the kidney has what may be termed a mesonephron and can be displaced in any direction from its proper site, the floating kidney.

Etiology.—In rare cases the condition is congenital. The acquired form is much more frequent in women, doubtless owing to the influence of tight-lacing and in some cases the relaxation produced by repeated pregnancies. Falls or heavy lifting may be the cause. Owing to its relation to the liver and consequent depression with every respiration the right kidney is much more frequently affected. The condition is often part of the general displacement of abdominal viscera, known as enteroptosis.

Symptoms.—(1) There may be none. The condition is often discovered as an incident of the physical examination. (2) A feeling of dragging or weight in the right side of abdomen or in the back may be complained of. (3) Reflex symptoms of greater or less severity may be excited. (a) Nausea or vomiting, indigestion or constipation may be caused by a floating kidney. These symptoms are usually intermittent, and are increased by influences affecting the strain produced by the displaced organ, such as walking or hard work. (b) Dietl's crises. In some instances the patients suffer from intermittent attacks of sudden pain in the abdomen, with chill, fever, nausea and vomiting, as in renal or biliary colic. These attacks are attributed to kinking of the ureter, and are sometimes followed by the passage of unusual quantities of urine. Like attacks may also be induced by over-indulgence in food and drink. During the attacks the urine may contain blood, or albumin and casts, or urates in abundance. The kidney may be enlarged and tender to pressure. (c) Manifestations of neurasthenia or hysteria may be caused by the presence of a floating kidney. Such cases are rare. In most instances the movable kidney is an accidental accompaniment of the general condition.

Diagnosis.—This must be based upon the identification of the mass by its position, size, form and consistency. It must be remembered that the kidney may be met with in any part of the abdomen or pelvis.

Treatment.—Many of the patients have no symptoms and require no treatment. Most of the cases with symptoms can be treated satisfactorily by an abdominal belt with a pad to support the kidney. Various corsets have also been invented for this purpose. Nephrorraphy may be required in the more extreme cases, or where the simpler measure fails. Nephrectomy is contemplated only when all other measures fail, or the mobility is complicated by serious disease of the kidney.

CONGESTION OF THE KIDNEY

Acute or Active Congestion.—An acute hyperemia of the kidney occurs in the early stages of acute nephritis. The etiology, pathology, symptoms, and treatment are those of acute Bright's disease, and there is at present no advantage in attempting to separate the two conditions.

CHRONIC OR PASSIVE CONGESTION results (1) from disease of the heart or lungs resulting in failure of the return circulation in the inferior vena cava; (2) from compression of the renal veins by tumors, including the pregnant uterus, or ascitic fluid. The kidneys are large and firm, the capsule strips easily, the cortex is deeply congested and bleeds freely on section. The markings are exaggerated. Microscopically the veins are engorged, there is some increase of fibrous tissue, some sclerosis of the glomeruli and degeneration of the tubular epithelium, a chronic diffuse nephritis. The urine is scanty and of high specific gravity, contains a slight or moderate amount of albumin and casts, possibly blood. Otherwise the symptoms are those of the primary disease. Uremia is rare.

TREATMENT must be directed to the cause.

ANOMALIES OF THE URINARY SECRETION

Anuria.—Suppression of the urine may result from (1) congenital absence of the kidneys. (2) Acute or chronic nephritis. No urine may be secreted for many hours. This occurs especially in severe acute infections, such as cholera, or yellow fever, and in the terminal stages of chronic nephritis. (3) Obstruction of both ureters by calculi, or of a single ureter, in case there is only one, or the removal of an only kidney. (4) Collapse following severe operations. (5) The passing of a catheter. (6) Hysteria.

SYMPTOMS.—In some cases symptoms are long delayed. With complete anuria patients may live for one or two weeks. In other cases, especially in conditions producing acute nephritis, uremic symptoms appear early and the condition is quickly fatal.

TREATMENT.—The obstructive cases require operation. In other cases, cupping or poultices over the loins, colonic irrigation with large quantities of hot salt solution, sweating by hot packs and pilocarpine given hypodermatically, and the free drinking of water may be effective.

Hematuria.—Blood may appear in the urine from various causes. (1) General diseases: (a) Diseases associated with grave changes in the blood, pernicious anemia, leukemia, scurvy, purpura, malaria, and possibly other acute infections. (b) Diseases causing renal congestion, such as chronic endocarditis or myocarditis, or obstructive lesions of the lungs, such as interstitial pneumonia. (2) Local conditions: (a) Trauma of any part of the genito-urinary tract. (b) Acute or chronic nephritis, or infarction of the kidney. (c) Calculi in kidney, ureter, or bladder. (d) New growths of the kidney or bladder. (e) Tuber-

culosis of the kidney or bladder. (f) Acute inflammations of the pelvis of the kidney, bladder, or urethra, such as occur in pyelitis, cystitis, or urethritis. (3) Parasites, such as the Bilharzia hematobia.

DIAGNOSIS.—Blood in the urine tinges it dark red or black. It can be distinguished from hemoglobinuria only by the demonstration of the corpuscles under the microscope.

TREATMENT must vary with the cause. Quiet is the one measure generally applicable.

Hemoglobinuria.—Whenever rapid hemolysis takes place in the body, hemoglobin may be excreted unchanged in the urine. This may follow transfusion, the foreign blood being broken up. It may result from the action of certain poisons, such as potassium chlorate, arsenurated hydrogen, or toluylendiamin. Infections such as malaria may be the cause, and hemolysis has also followed extensive burns or exhausting exercise.

PAROXYSMAL HEMOGLOBINURIA is a clinical condition without definite pathology or etiology. Malaria and syphilis are adduced as causes. Often no cause can be found. Exposure to cold, or severe exertion may be the immediate cause of the attacks which characterize the affection. These attacks resemble the malarial paroxysm, beginning with a chill, followed by fever, and sweating. In these attacks extensive hemolysis occurs from unknown causes. During or after the chill the patient passes blood-tinged urine and continues to do so for several days. He is then well for an indefinite period. The urine is either lightly or heavily colored by hemoglobin. The microscope shows no red blood cells. The urine regularly shows albumin derived from the blood. The presence of hemoglobin may be demonstrated by the spectroscope. The nature of the affection is unknown. It is supposed to be related to Raynaud's disease, symptoms of which sometimes appear with hemoglobinuria.

Albuminuria.—The presence of albumin in the urine was first noted in Bright's disease. For a long time its presence was regarded as indicative of nephritis. Accumulating evidence, particularly the use of the more delicate tests for albumin, has shown that it may be found in the urine of persons giving no evidence of Bright's disease or any other renal lesion. It may appear in the urine of persons apparently in perfect health, but it is generally agreed that albumin is not excreted by an entirely normal kidney. Practically, we know that some persons can go on for years excreting albumin in small quantities either constantly or intermittently without developing other signs of nephritis, or other renal lesion. We therefore classify the condition as

1. Albuminuria without gross renal lesions, including (a) Physiological albuminuria, in which albumin appears transiently or constantly in the urine of apparently healthy persons. Various conditions may lead to the excretion of albumin, such as the ingestion of large amounts of albuminous foods, especially eggs, severe exertion, or cold bathing.

In other persons no cause can be assigned. The condition has been termed cyclic, paroxysmal, intermittent, or orthostatic albuminuria, as the individual case suggested. (b) Hemic albuminuria. Grave anemia from any cause may lead to mild albuminuria. (c) Febrile albuminuria. Fever of any kind may cause the presence of albumin in the urine. In fever the urine is regularly scanty, of high specific gravity, contains a trace of albumin, and often hyaline and granular casts. With the subsidence of the fever the urine becomes normal again. A large percentage of all fever cases show these changes. They are attributed to the action of toxins. In some cases there are doubtless organic changes, such as the cloudy swelling seen post-mortem in fatal cases. In other cases it is possible that the kidneys remain normal. (d) Nervous albuminuria. After epileptic seizures, in apoplexy, tetanus, exophthalmic goitre, or after injuries to the brain, albumin may appear in the urine.

2. Albumin with definite kidney lesions. (a) Congestion, acute or chronic, of the kidney. (b) Nephritis, acute or chronic, of any type. (c) Suppurative inflammation of any part of the urinary tract. (d) Tumors of the kidney, ureter, or bladder. The significance of the presence of albumin in the urine will be determined by the conclusion as to its cause. It is usually a symptom of importance, but in some cases exists for years without apparent harm.

Pyuria.—Pus in the urine may come from (1) Suppurative inflammation in the genito-urinary tract; (a) urethritis, most often gonorrheal; (b) cystitis or prostatitis; (c) pyelitis; (d) pyelonephritis. (2) Abscesses or suppuration in other parts discharging in the urine; (a) leucorrhea; (b) pelvic, perinephritic, or appendical abscesses.

Pus must be identified by the microscope. With pyuria the urine is often alkaline, but may remain acid.

Chyluria.—The urine may present a milky appearance from admixture with chyle. The condition may arise from infection with filaria, or in some cases by chance communication of lymphatic vessels with the pelvis of the kidney or ureter. Chylous urine is frequently pink from the presence of blood. Under the microscope the fat globules and possibly red blood cells are seen.

Lithuria.—The excretion of excessive amounts of uric acid and urates is thus designated. Both these substances are end results in the metabolism of nitrogenous foods, being derived from the nucleo-proteids. Their elaboration in the body runs parallel with the formation of urea, but is not identical with the latter. Uric acid may be derived either from the nucleo-proteids of the food, or those of the body itself, and is thus distinguished as exogenous or endogenous. The amount excreted must therefore depend in part upon the diet, in part upon the breaking down of body cells. We have ordinarily no means of determining how much of the uric acid appearing in the urine is derived from each of these sources. The problem of interpreting variations in the excretion of uric acid is therefore so complex that we can at present make prac-

tically no deductions of clinical value from observations of the excretion in individual cases. Exhaustive studies of the metabolism of gout have failed to demonstrate any constant excess or deficiency of the uric acid of the urine. At most we have learned that just before an attack the excretion of uric acid is diminished, while as the attack subsides it is increased. The diagnosis of gout cannot be made from the study of the urinary conditions alone.

Deposition of uric acid and urates in the urine is a common occurrence, the sediment having a pale amorphous appearance if the urates are in excess, or appearing as minute reddish crystals, the "brick-dust" sediment, if uric acid crystals are present in quantity. Such precipitation of urates and uric acids results from changes in the temperature, specific gravity, reaction and content of other substances, especially the chromogen of the urine. Such deposits do not, therefore, necessarily mean increased elimination.

In brief, excretion of uric acid and urates will depend chiefly upon the amounts ingested and the body metabolism. Sedimentation of uric acid and urates will depend in part on the presence of excessive amounts, but more largely upon the other characters of the urine. It is a common phenomenon in all febrile cases. Recognition of these facts should prevent us from attaching too great value to the appearance of sediments containing uric acid, or to estimations of the amount to be found in the urine of any individual patient.

Oxaluria.—The deposition in the urine of crystals of calcium oxalate is of importance because of the frequency with which this substance forms concretions in the kidney or bladder. Calcium oxalate may be derived from food or result from the metabolic activities of the body. The exact origin of the endogenous portion is not known, and therefore the significance of an increased output, apart from the question of intake, is still undetermined. The effort to lessen the output must be directed to the removal from the diet of the substances richest in this salt, such as rhubarb, spinach, and tea.

Cystinuria is a very rare condition in which the colorless hexagonal crystals of cystin appear in quantity in the urine. They may be the basis of calculi. Excessive excretion belongs to certain individuals and often persists throughout life. The disposition may be hereditary. The cystin results from a defective metabolism of protein.

Phosphaturia.—Phosphates are normally present in the urine. They may be deposited by reason of excessive excretion, or, when normal in quantity, because of a change of the normally acid urine to an alkaline reaction. Only to the former condition should the term phosphaturia be applied. True phosphaturia is often associated with neurasthenia or disturbances of the sexual organs. The phosphates excreted are mainly those of sodium, potassium, calcium, and magnesium. Of these the sodium and potassium salts are the more easily soluble. By reason of this fact an excessive excretion of Ca and Mg is sometimes the explanation of a phosphaturia, these bases uniting with phosphoric

acid to form the less soluble salts and also diminishing the amounts of sodium and potassium phosphates upon which the acidity of the urine depends.

Indicanuria.—The intestinal bacteria break up the proteids of the food and produce indol. The indol is taken into the circulation and oxidized, probably in the liver, into indoxyl, which combines with sulphuric acid to form the so-called ethereal or conjugate sulphates. This indoxyl when treated with strong oxidizing agents is broken up with the formation of indican or indigo. The common test for indican consists in adding to 5 to 10 c.c. of the urine an equal amount of Obermeyer's reagent, composed of strong hydrochloric acid (specific gravity 1019) containing two parts per thousand of ferric chloride. One to two c.c. of chloroform is then added and the whole gently mixed by repeated inversion of the tube. If indican is present, it will be taken up by the chloroform, which on allowing the mixture to settle sinks to the bottom and appears stained more or less deeply blue. A trace of indican may be present in health. In large amounts it is abnormal. It is most abundant in cases where intestinal fermentation is most active, such as typhoid fever, intestinal obstruction, and the like. Its presence has most value in the milder functional intestinal disorders, often described as "biliousness," where the occurrence of excessive indican formation suggests the desirability of reducing the proteid intake and of instituting measures for the lessening of intestinal fermentation.

UREMIA

Definition.—Uremia is the condition of intoxication developed in acute or chronic nephritis. The cause is unknown. (1) It was long attributed to the retention of substances normally excreted. Urea, the potassium salts, creatin, and uric acid have been successively named as the harmful substances, but proof of their relation to the intoxication has never been given. (2) An internal secretion, like that of the thyroid, has been assumed to explain the phenomena, but this remains a pure hypothesis without evidence to support it. (3) Still another theory attributes the symptoms to the effects of neurolysins produced in the diseased kidneys upon the nerve centers. The fact remains that at present there is no satisfactory explanation of the condition known as uremia. The greatest difficulty in the understanding of the matter arises from the fact that the phenomena of uremia are not those of anuria, and that patients have been known to survive periods of anuria of two weeks, or in one case nineteen days.

Symptoms.—These are developed either swiftly or slowly. In some cases, especially in acute nephritis, the symptoms come so swiftly that the cases are properly termed fulminant, in other cases the phenomena of uremia are gradually developed in the course of years. Commonly, cases are classified as acute or chronic, according to their rapidity of evolution. (1) Headache is a frequent and often an early symptom.

It is usually occipital, and may be either mild or agonizing. (2) Ocular symptoms are common. Impairment of vision may result from optic neuritis, but apart from demonstrable changes in the retina, vision becomes dim or total blindness supervenes. (3) Dyspnea is frequent and severe, and may be constant or paroxysmal. It is regularly worse at night, and orthopnea is not uncommon. In the severe types Cheyne-Stokes respiration is often seen. (4) Nausea, vomiting, and diarrhea are common and sometimes the only symptoms. The vomiting may be only occasional, sometimes it is persistent. Diarrhea, once it appears, is likely to be persistent and intractable. In some cases marked organic lesions, such as membranous colitis, are found post-mortem, but in others the intestines appear normal. (5) Muscular twitchings are usually present. They involve the muscles of the face or other part, or the whole body. (6) Delirium, coma, or convulsions develop in the severer cases. The delirium may be mild, wandering, or maniacal in type. The coma is usually profound with complete insensibility and loss of control of the sphincters. The pupils during coma may be normal, or contracted, or even dilated. Contraction is the more frequent. The convulsions are epileptic in type and may be either local or general. Once developed they are usually repeated at short intervals, the patient remaining comatose between them. Monoplegia or hemiplegia may develop during or after a convulsion and may persist for some time, but such paralyses regularly clear up in those who survive. (7) The urinary symptoms are not constant. The urine usually shows on examination the characters belonging to a parenchymatous or interstitial nephritis.

Diagnosis.—The recognition of milder degrees of uremia often depends upon the thorough investigation of patients complaining of minor ailments such as headache, neuralgic pains, insomnia, occasional nausea or vomiting. Careful examination of the patient may develop the characteristic conditions of a chronic nephritis, the findings being confirmed by the urinalysis. Routine examination of the urine, both chemically and microscopically, in all patients, more especially those over forty, is the only safe-guard against error. Uremic coma in a patient seen for the first time is difficult of diagnosis. It is most likely to be confounded with coma due to alcoholism, cerebral hemorrhage, opium-poisoning, or diabetes. In uremic coma the unconsciousness is profound, the pupils are equal, either dilated, normal or contracted, there is no deviation of the eyes. The breath has a peculiar urinous odor. Usually there is no paralysis, but a monoplegia or hemiplegia may be present, which cannot at the moment be distinguished from paralysis due to cerebral hemorrhage. If the patient survives, the paralysis gradually disappears. Lumbar puncture gives normal cerebrospinal fluid, usually under increased pressure. The urine, withdrawn by catheter, contains albumin, usually in considerable quantity and abundance of casts. The specific gravity may be high or low. There may be definite suggestions of nephritis in thickened

arteries, increased blood pressure and hypertrophy of heart or in the presence of edema and anemia.

Alcoholic coma is less profound. The patient can usually be roused, either by pressure over the supra-orbital nerves, or by the policeman's more crude, but satisfactory method of beating the soles of the shoes with his stick. The face is flushed, the breath heavy with the odor of alcohol, the pulse rapid and bounding, with no change in the pupils, no paralyses. The possibility that alcohol may have been given to a patient suffering from another cause must be remembered.

Opium-poisoning is marked unmistakably by the profound coma, pin-point pupils, and slow respiration, the rate falling to 12 or 8 or less in the minute.

Diabetic coma usually occurs in persons who are emaciated by their disease; the breath has an ethereal odor, ascribed to acetone; the skin is dry and harsh; the urine is of high specific gravity and contains sugar.

In protracted cases uremic coma has been confused with the stupor of typhoid fever, cerebrospinal fever, or tubercular meningitis.

The TREATMENT of uremia is included in that of chronic nephritis.

FUNCTIONAL EFFICIENCY OF THE KIDNEYS

In studying the effects of nephritis we much concern ourselves not only with the presence of abnormal elements such as albumin and casts in the urine, but with the problem of the influence of disease upon the functions of the kidneys as the chief excretory organs of the body. For many years the specific gravity of the urine and the total quantity for 24 hours furnished the only standard for such judgments. The normal quantity of urine for 24 hours is 40 or 50 ounces, the normal specific gravity from 1015 to 1025. As the quantity increases the specific gravity normally falls. The rough rule has been established that the product of the last two figures of the specific gravity by 2 or 2.33 (Häser's factor) gave the solid excretion in grams, and if this exceeded 35, the urinary excretion could be regarded as satisfactory. Many influences, such as the diet and fluid intake, exercise, the activity of the skin and bowels, modify the factors in this method of computation and affect the result. The rule expresses only the minimum standard of efficient excretion and must be loosely applied in any individual case.

Efforts have been constantly made to obtain a more exact standard. This was sought in the determination of the percentage and daily output of urea, because of the assumed relationship of the retention of urea to the symptoms of uremia. The information thus obtained has not proven of practical value, and the effort to judge of the efficiency of the kidneys by computation along this line has been abandoned.

More recently estimation of the freezing-point of the urine after the method of Koranyi, or calculation of the electrical conductivity of the urine, has been proposed as a standard of renal efficiency, but after

protracted trial these procedures have been found no more satisfactory and are no longer employed.

At the present time the attention of students of this subject is directed not to the estimation of the total excretory activity of the kidneys so much as to their ability to excrete individual substances, and more especially sodium chloride and water.

Chloride Excretion.—It has been clearly shown that diseased kidneys vary greatly in their ability to excrete sodium chloride. The normal chloride excretion varies between 10 and 15 grams per day. In certain cases of nephritis the total may fall to 1 gram or even less. In such cases under ordinary conditions there results a marked retention of sodium chloride in the blood and the tissues of the body. Upon such salt retention edema in some cases depends, and the amount of edema is found to vary with the retention or excretion of the chlorides. The value of a salt-poor or relatively salt-free diet in these conditions has been established and restriction of the salt intake is now recognized as a potent therapeutic measure not only in nephritis but in other conditions marked by serous effusions.

The study of the chloride intake and output in connection with the water intake and output and the weight of the patient has therefore been established as a measure of value both in the diagnosis and prognosis of nephritic lesions.

If the kidney still retains the ability to excrete salt normally or nearly so, the reduction of nephritic edema may easily be brought about by the employment of a diet having a low salt value.

Therein lies in part at least the explanation of the efficacy of a milk diet in nephritis. If, on the other hand, the ability to excrete salt is impaired or nearly lost the problem of reducing nephritic edema is much less easily solved.

Experimental evidence indicates that salt excretion is most reduced in processes involving the epithelium of the tubules, while vascular lesions affect it slightly or not at all. Salt retention should, therefore, be characteristic of nephritis affecting especially the epithelium (parenchymatous nephritis), while it should be less marked in those types of nephritis involving especially the glomeruli and sparing the tubules (interstitial nephritis). But we are not yet in a position to apply these propositions as criteria to determine the anatomical type of nephritis with which we may be dealing.

Water Excretion.—In like manner in certain cases of nephritis the power to excrete water seems to be impaired or preserved, independently of the chlorides. For this reason the free administration of water may be beneficial in some cases of nephritis and harmful in others. Impairment of ability to excrete water indicates lesions particularly affecting the glomeruli, but in certain cases of nephritis in which the glomeruli are extensively damaged the function of water excretion seems to be taken over by the epithelium of the tubules and carried on so actively that even an excess of water may be excreted (interstitial nephritis).

The study of the influence of the free administration of water or its restriction upon the urinary secretion, the edema, and the weight of the patient is of great value in determining the proper procedure. The normal kidney will excrete water freely up to 100 or 120 ounces in 24 hours, and the higher the amount, as a rule, the larger the proportion of the water excreted under ordinary conditions by the kidney. If the kidney be seriously diseased, the power of excreting water may be notably impaired, and no diuresis follow the addition of 50 or 60 ounces of water to the fluid intake. It is not uncommon to find the total water excretion of the kidneys limited to 10 ounces or less per day. Under such conditions the free administration of water may be harmful, and the other avenues of eliminating fluid must be resorted to. On the other hand, if the water excretion is still free it may be advantageous to give large amounts.

Excretion of Other Substances.—Efforts to test the functional capacity of the kidneys by the administration of methylene blue, salicylic acid, or potassium iodide by mouth, or phenolsulphonephthalein hypodermatically have been made, but the value of these methods in calculating the efficiency of the kidneys has not yet been established. They have, however, been employed successfully in comparing the relative efficiency of the two kidneys in cases in which one only is impaired.

ACUTE NEPHRITIS

(Acute Bright's Disease)

Definition.—An acute inflammation of the kidney, involving to some extent both the interstitial and parenchymatous tissue.

Etiology.—Acute nephritis occurs at all ages, but especially in childhood. (1) It may arise without apparent cause, a primary nephritis. It is more often due to the following influences: (2) Exposure to cold or wet. (3) The poisons of the acute infectious diseases, especially scarlet fever. (4) Alcoholic excesses predispose to the disease. (5) Pregnancy. The nephritis complicating pregnancy is best classified as an acute nephritis, though presenting many points peculiar to itself.

Morbid Anatomy.—The gross appearances are not constant. The kidneys are usually somewhat enlarged, softer than normal, the capsule free, the surface smooth and either congested or pale, the section cloudy, the markings being less distinct than normal. Microscopically the lesions of acute nephritis are of very irregular distribution and in different cases affect different parts of the tissue, the glomeruli, the tubules, the interstitial tissue especially. Thus glomerular, tubular, and interstitial types of acute nephritis have been described by different authors, depending upon the part found most affected. It is, however, impossible to distinguish these groups clinically. The lesions found in the kidney tissue may be grouped under the headings of congestion, degeneration, exudation. The congestion varies greatly in degree and

in distribution. It may be absent entirely. Degenerative changes are always present, especially in the epithelium of the tubules. Different parts of the tubules are variously affected, the convoluted tubes usually showing the most marked lesion. The epithelium becomes granular or fatty, or may break down completely, and the nuclei may disappear. The glomeruli show similar changes. An exudation of serum, fibrin, and leukocytes or red cells may be found sometimes in the tubules, sometimes in the glomeruli, sometimes in the interstitial tissue, rarely in all parts of the kidney in any one case. The amount of such inflammatory exudate varies greatly. In some it is so slight as to be seen with difficulty. In other cases it is abundant, the tubules, glomeruli, and even the interstitial tissue showing leukocytes and other inflammatory products. Councilman has described a special variety of acute nephritis in children in which the exudate in the kidney consists almost wholly of plasma cells.

Symptoms.—The onset is either (1) abrupt or (2) insidious. (1) In the abrupt cases the invasion is marked by malaise, fever, headache, possibly nausea and vomiting, and some edema of the face and extremities, and possibly the abdomen and chest. (2) In the insidious cases the invasion is very gradual, marked by a little malaise, lassitude, and pallor. On examination of the urine in either case the characteristic changes are found. The urine is scanty in amount, high colored, possibly bloody, of high specific gravity, with albumin in abundance and microscopically many casts, hyaline, granular, epithelial and sometimes blood-casts, often red and white blood cells and much granular detritus. There may be complete suppression of the urine for many hours, possibly 1 or 2 days. Once the disease is established the course varies. (1) Some patients recover within a week or two. (2) Others develop severe uremic symptoms, headache, dyspnea, delirium, coma, possibly convulsions, and die within a few days. (3) The majority of the cases run a protracted course, the symptoms already enumerated varying from time to time, until ultimately they recover, or the condition of chronic nephritis is developed, usually of the parenchymatous, but sometimes of the interstitial type. In these cases the marked symptoms are (1) edema of the dependent parts, especially the legs and scrotum, often a general anasarca involving the pleura and peritoneum. The anasarca may be very persistent. (2) Anemia is always marked. (3) Loss of appetite, nausea, and occasional vomiting. (4) Variable uremic symptoms, headache especially. (5) Dyspnea is absent, except on exertion or when the chest is full of fluid. (6) The urine remains scanty and of high specific gravity, with albumin and casts, but the albumin gradually lessens and the casts likewise. Blood usually disappears after the acute stages. (7) In many cases the pulse tension is high, the arteries gradually thicken, the heart hypertrophies, and the vascular conditions characteristic of chronic nephritis develop.

Diagnosis.—This rests on the combination of symptoms and urinary findings. The chief source of confusion is the febrile albuminuria. In

any febrile disease the urine may show a trace of albumin, with hyaline and granular casts. As the fever subsides these disappear. Such cases should not be classed as acute nephritis.

Treatment.—The essential point is as complete rest for the kidney as possible. This is secured by (1) rest in bed; (2) a fluid diet, especially milk or the low-salt diet (p. 593), is indicated; (3) free purgation by salts; (4) hot packs or hot air baths to secure free sweating daily or every other day. Pilocarpine may be given for the same purpose, gr. 1/12–1/10 for an adult. (5) Water should be given freely as a diuretic if freely excreted. The anasarca sometimes requires further interference. Effusions in the peritoneum or pleura should be withdrawn when large enough to embarrass respiration by pressure. They usually return promptly. The legs and scrotum may be punctured with a needle or Southey's tubes may be employed, but much the best plan is to make several long incisions, under cocaine, through the skin. All wounds must be treated aseptically. For anuria, poultices may be applied over the loins, and rectal irrigations of normal salt solution heated to 105° to 110° F. Purgatives and sweating may be employed. For severe uremic symptoms venesection is indicated if the pulse tension is high. Nitroglycerine may be given instead. Chloral alone or chloral and sodium bromide may be given by the mouth or rectum. Morphine may be employed hypodermatically to prevent or mitigate convulsions. Chloroform may be used for the same purpose. Lumbar puncture has been employed for the reduction of intracranial pressure, apparently with good results. While these palliative measures are being employed, every effort must be made to promote diuresis and lessen the work of the kidney by the measures already suggested. As the condition progresses the diet must be enlarged by the addition of cereals, fruits, and vegetables. The anemia should be treated by iron, and the treatment gradually modified to that of a chronic nephritis.

CHRONIC NEPHRITIS

The pathological anatomy and the clinical history of cases of chronic nephritis vary greatly in different patients. Numerous classifications, all more or less unsatisfactory, have been made and cast aside. We have come to accept two fairly definite types, the parenchymatous and interstitial, named from the predominant feature of the pathological change in each, although it is recognized that, strictly speaking, the terms are misleading, for in every chronic nephritis all parts of the kidney, tubules, glomeruli, interstitial tissue, are involved to some extent. We also know well that many cases fall neither in one group nor the other, but present a mixed and unclassifiable picture. Nevertheless these two fairly well differentiated types at present serve as the most satisfactory categories under which to describe the phenomena of chronic nephritis.

CHRONIC PARENCHYMATOUS NEPHRITIS

(Large White Kidney)

Definition.—A chronic inflammation of the kidney affecting especially the parenchyma of the organ.

Etiology.—(1) It is often the sequel of an acute nephritis. (2) The excessive use of alcohol, syphilis or tuberculosis predisposes to it. (3) In the great majority of cases the disease appears without recognizable cause.

Morbid Anatomy.—Many varieties of chronic parenchymatous nephritis are described, such as the large white kidney of Wilks, the large red, and the small white. Neither the size nor the color of the kidney is of great importance, but these three types may be taken as representing the variations commonly seen. The large white kidney described by Wilks is much larger than normal, the two organs sometimes weighing as much as 25–30 ounces (800–900 gms.). (See Fig. 25.) They are firm, the capsule is free or but slightly adherent, the surface is pale gray or slightly yellow, the cortex is thick, the markings are much obscured. Microscopically the lesions are found irregularly distributed. There is a moderate increase of connective tissue, which in places shows foci of round-cell infiltration. The striking changes are in the tubules and glomeruli. In the tubules there is marked degeneration of the epithelium, often with desquamation. The lumen of the tubes often shows casts. The convoluted tubules are most affected. The glomeruli are variously changed, some larger, others smaller, some completely atrophic. In general, Bowman's capsule is thickened and its epithelial covering sometimes increased. Over the tufts there is an increase of cells. Many of the tufts show hyaline degeneration, and some are converted into fibrous tissue. The walls of the blood-vessels, especially the smaller, may show thickening.

The large red kidney presents no material difference except in color. The small white kidney is regarded as a late stage of the large white, in which there has been a marked increase of connective tissue with subsequent contraction, reducing the size of the kidney. These kidneys are smaller than normal, firmer, the capsule is more adherent, the surface is pale, the cortex of normal width or less, the markings indistinct. Microscopically the lesions have the same character as in the large white kidney, except that there is more fibrosis, both in the interstitial tissue and the glomeruli.

ASSOCIATED LESIONS.—In fatal cases fluid is found in the serous cavities, especially the pleura and peritoneum. There may be an acute pleurisy, pericarditis, or peritonitis, representing a terminal complication.

Symptoms.—(1) When the disease is a sequel of an acute nephritis, anemia, edema, dyspnea, and the urinary conditions persist. (2) In other cases the invasion is insidious. (a) Anemia develops early. (b) Gastric symptoms are usually marked; loss of appetite, possibly

nausea and vomiting. The bowels are constipated. (c) Edema appears, in the ankles first, or in the face, later fluid is found in the peritoneum, pleura, or pericardium. (d) The urine shows a reduced quantity, of high specific gravity, containing large quantities of albumin and many casts. Blood cells may appear in the urine, but are not so frequent as in the acute form. (e) Drowsiness and headache are common, but

Fig. 25.—The large white kidney: cortex of normal thickness and its markings fairly preserved.

the severer uremic manifestations, such as muscular twitchings, delirium, coma, and convulsions, are comparatively infrequent. (f) The heart may hypertrophy and the vessels show sclerosis, but these changes are more common in the interstitial nephritis. They are therefore apt to be associated with the small white kidney rather than the larger forms.

Course and Prognosis.—Once the disease is well established it is

rarely cured. It runs a chronic course and the patients, after months or years, succumb either to the general anasarca and edema of the lungs, or to a complicating inflammation of one of the serous membranes, or to uremic convulsions. During the course of chronic parenchymatous nephritis exacerbations closely resembling acute nephritis are frequent.

Treatment.—The main lines of treatment are those of acute nephritis. The patient must lead a quiet life; if edema is present he had best be kept in bed or in a chair. Milk should form the basis of the diet, with the addition of cream, cereals, and syrup, as in Von Noorden's diet. Water should be given according to the ability of the kidney to excrete it. Diuretics, the acetates of potassium and sodium, diuretin and the like, may be tried at times, but the results are usually disappointing. Anemia is treated by the administration of iron. Basham's mixture is usually chosen. The bowels must be kept open and the skin active. When possible, life in a mild, uniform climate, such as that of Southern California, is desirable. Anasarca must be treated as in acute nephritis. When all other methods fail, decapsulation of the kidneys may afford relief and indefinitely prolong the patient's life.

CHRONIC INTERSTITIAL NEPHRITIS

(Granular or Contracted Kidney)

Definition.—A chronic nephritis involving especially the interstitial tissue.

Etiology.—Three forms of contracted kidney are recognized. (1) A secondary form which follows or develops from a chronic parenchymatous nephritis. In this type the etiology of chronic parenchymatous nephritis applies. (2) The arteriosclerotic kidney, in which the disease is part of a general arteriosclerosis, and is due to the causes producing the latter. (3) A primary type, often developing without definite cause. A number of factors are believed to influence the production of such nephritis: (a) the excessive use of alcohol; (b) faulty digestion or metabolism due to overeating; (c) gout or diabetes; (d) acute infectious diseases, especially scarlet fever, are sometimes followed by contracted kidney. The disease is common in people over 40 years of age, but is seen occasionally in earlier years, even in childhood.

Morbid Anatomy.—The kidneys are small, the two sometimes weighing only 50–60 grammes; they are hard; the capsule is tightly adherent, so that in stripping it off, bits of the cortex are torn away (see Fig. 26); the color is usually red; the surfaces very coarsely granular, and often marked by numerous small cysts, caused by obstruction of some of the tubules or glomeruli; the substance cuts with resistance; the cortex is thin, in places almost obliterated; the markings very indistinct (see Fig. 27). Microscopically the striking feature is the marked increase of connective tissue everywhere. In the dense strands of fibrous tissue the glomeruli and tubules appear

to be strangled. Some few are normal, most of them show pronounced changes. Many of the glomeruli are represented only by fibrous patches, many are very small with the capsule much thickened, the epithelium lining the capsule and covering the tuft much increased. Some few glomeruli appear distended (from obstruction). Similarly many of the tubules are obliterated, many are very small, some few are distended. The epithelium shows degenerative changes, but not so markedly as in the parenchymatous type, and is often simply flattened. Many of the cells are broken down or desquamated. The lumina of the tubes is often filled by casts. The blood-vessels are notably thickened, especially their middle coats. It must be understood that the description here given is that of the typical example. In many cases the changes are less marked and so closely resemble those of chronic parenchymatous nephritis that

Fig. 26. Fig. 27.

Fig. 26.—Small contracted kidney, showing the rough, granular surface, and at the upper pole a small cyst.
Fig. 27.—Section of the contracted kidney, showing the atrophic cortex with obliterated markings and cysts, and the fatty tissue about the pelvis.

it is impossible to say from the anatomical conditions alone in which class a given kidney belongs. Also, there are cases of chronic nephritis which with all the data known (history, urinalysis, gross and microscopic examination of the kidney) cannot be satisfactorily grouped under either of the classes here given, but must be designated simply as chronic nephritis.

Associated Lesions.—The heart is hypertrophied, the aorta may show atheroma, and the smaller vessels are thickened. The lesions of cerebral apoplexy, a frequent cause of death, may be found. Dropsical effusions may be present. Acute pleurisy, pericarditis, or peritonitis is occasionally seen.

Symptoms.—The invasion of the disease is so insidious that it is often not discovered till far advanced, or till some of the complications

bring about a careful examination; not infrequently the condition is discovered only at autopsy. The patient most often complains simply of not feeling well, or of headache, or insomnia, or dyspnea, or disturbance of digestion with loss of appetite and flatulence, or of polyuria. Examination then discloses: (1) Urinary condition. The urine is increased in quantity, 2000–4000 c.c. in 24 hours, the specific gravity is 1010–1015, a trace of albumin is present, and hyaline and granular casts. The solid constituents are diminished in proportion and their total daily elimination is low.

(2) The heart is hypertrophied, especially the left ventricle, the second aortic sound is exaggerated, the pulse tension is increased, sometimes enormously. The sphygmomanometer readings vary from 175 to 300 mm. of Hg. The arteries are thickened and more or less rigid.

(3) The retina may show a diffuse retinitis or scattered patches of hemorrhage and atrophy. The eye changes belong, as a rule, to the later stages.

(4) Edema or anasarca is rarely found until the heart fails under the increased peripheral pressure and congestion results.

Once the disease is established, the course is protracted and variable. (a) The patient may go on for many years with health slightly impaired, but suffering very little, life being ended by some intercurrent acute disease, especially pneumonia. (b) Severe uremic symptoms, headache, delirium, coma, or convulsions may be fatal at any period. Not infrequently the advent of coma or convulsions is the first sign of the disease to attract attention. Vision may be impaired or blindness result from the retinal changes. (c) Dilatation and failure of the heart with congestion and edema often mark the close of the disease. (d) Apoplectic attacks are frequent. A single attack may be fatal, or there may be a number. In some cases a transient monoplegia or hemiplegia occurs. Localized edema of the brain or spasm of the cerebral arteries has been suggested as the explanation of such temporary loss of power.

The BLOOD regularly shows a secondary anemia of moderate grade.

Diagnosis.—This must be based upon the presence of the urinary changes with the characteristic changes in the heart and arteries. The examination of the retinæ may be important. The possibility of latent interstitial nephritis makes necessary the examination of the urine in all patients, more especially those over forty years of age.

Prognosis.—Chronic interstitial nephritis may exist for many years without serious results. On the other hand, the disease is incurable. The changes in the heart and vessels are, as a rule, most important elements in the prognosis. The estimation of the blood pressure is helpful. Marked changes in the retinæ are signals of danger; with them patients rarely survive more than a year.

Treatment.—Except in the emergencies caused by sudden heart failure, or uremic crises, the treatment of chronic interstitial nephritis consists mainly in the regulation of the diet and life of the patient. (1) The diet should be of mixed composition and abundant enough

to maintain the nutrition of the patient. It should be determined by the appetite of the patient and his ability to digest rather than by theories as to what is indicated by the disease. Moderation is necessary. Alcohol in any form is withdrawn or restricted. Tobacco must be used sparingly, if at all. (2) Climate. In winter the milder temperatures of the Southern States, the Riviera, or Egypt, are sought. In summer some of the cooler resorts on the seashore or in the mountains of the North are recommended. Exposure to cold or wet is likely to induce attacks of bronchitis or pneumonia with serious results. (3) Exercise must be regulated and hard work of body or mind given up or restricted. (4) The bowels must be kept open, by laxatives if necessary, and the activity of the skin promoted by warm baths. Cold bathing is forbidden. (5) The regulation of the blood pressure must be carefully considered. If very high, over 200 mm., and especially if accompanied by evidences of cardiac failure, it should be reduced. In milder degrees nothing more than the measures already advised is required, and even when apparently excessive it may be wisest let alone, since it is apparently a protective measure. Severe symptoms, such as headache, convulsions, or dilatation of the heart, may demand its reduction. For this purpose rest in bed and a fluid diet are often effective. In emergencies bleeding may be resorted to. Vasodilators are employed, most often nitroglycerine in doses of 1/100 grain every two hours, increased till an effect is produced. The nitrites of sodium or potassium are sometimes used instead. Chloral is often valuable, allaying nervous symptoms and relieving excessive tension as well. (6) Uremic symptoms call for active treatment. The milder are met by free purgation, sweating by hot packs or baths, colonic irrigations, and the use of nitroglycerine. Convulsions demand bromide and chloral, by mouth or rectum, morphine hypodermatically or chloroform. Venesection is employed, especially if the tension is high, and sometimes followed by an infusion of salt solution, which while diluting the blood does not materially raise the blood pressure. Lumbar puncture has also been employed in these cases with good effect, if the quantity of cerebrospinal fluid is increased and under high pressure.

AMYLOID DISEASE OF THE KIDNEY

Etiology.—Amyloid degeneration of the kidney is always part of a general process involving the liver, spleen, and other organs as well. The causes are (1) prolonged suppuration, such as occurs in chronic empyema, osteomyelitis and the like; (2) late tuberculosis, especially the forms involving the lungs and bones, doubtless here also suppuration is the more direct cause; (3) tertiary syphilis.

Morbid Anatomy.—The kidney is large, the capsule free, the surface smooth and pale, the section showing a peculiar smooth, glistening surface, which on being tested with iodine becomes mottled with patches of mahogany-brown, representing the scattered areas of amyloid sub-

stance.   In rare instances the kidneys are small, hard, and granular, as in interstitial nephritis.

Symptoms.—The patients are pale and usually edematous.   The urine varies greatly in specific gravity and amount.   It contains large quantities of albumin and casts of all kinds.   Waxy casts may be found. The liver and spleen are enlarged, palpable, with smooth, firm surface, and sharp, hard edge.

The DIAGNOSIS must rest upon the etiology, and the combination of the urinary conditions with evidence of similar changes in the liver and spleen.

The PROGNOSIS is usually very bad.   When the diagnosis is fully established, the prospect of life is limited to a few months.

Treatment.—The underlying condition must be treated.   The amyloid degeneration is managed on the principles of chronic parenchymatous nephritis.

CYSTIC DISEASE OF THE KIDNEY

(1) Cysts develop in the kidney as the result of obstruction in many cases of chronic nephritis.   They are small and have no clinical
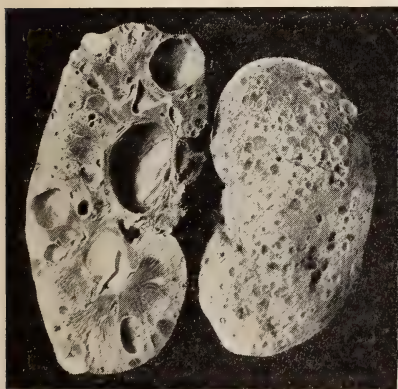
Fig. 28.   Fig. 29.
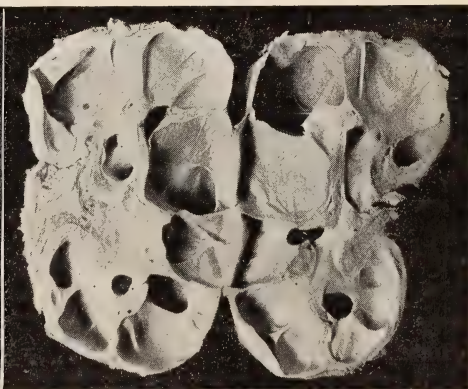
Fig. 28.—The cystic kidney, showing multiple cysts with partial preservation of the renal tissue.
Fig. 29.—The cystic kidney, showing the kidney converted into multiple cysts with thin walls.

importance.   (2) Solitary cysts of some size may occur and may reach such size as to require operation.   They are exceedingly rare.   Hydronephrosis from obstruction of the ureter might be classed under this heading.   (3) True cystic disease of the kidney is congenital, and affects both kidneys.   The kidneys, by reason of some unexplained developmental defect, are converted into a series of cysts of varying size, with little or no renal tissue left between them (see Figs. 28 and 29).   The cysts contain dark fluid, which shows albumin, blood crystals, cholesterin, and fat droplets, rarely urea.   The kidneys may be

so large and so placed in the pelvis as to obstruct labor; in the adult they may weigh 5 to 6 pounds.

The SYMPTOMS are those of chronic interstitial nephritis, with bilateral tumors in the loins, sometimes large enough to almost fill the abdomen. The condition is regarded as congenital, but is often not recognized till adult life.

The only treatment is that of nephritis.

PYELITIS

(Pyelonephritis. Pyonephrosis. Surgical Kidney)

Definition.—An inflammation of the pelvis of the kidney, either catarrhal or suppurative. In the latter the process regularly extends into the substance of the kidney and constitutes a pyelonephritis, or pyonephrosis.

Etiology.—The disease is of bacterial origin. The bacteria reach the kidney through open wounds, or from the bladder through the ureter (ascending infection), from the blood, or directly from the overlying colon. Any influence which lowers the vitality of the patient, such as the acute infectious diseases, or any condition causing stagnation of urine, such as phimosis, enlarged prostate, tumors of the bladder, calculi in the pelvis of the kidney or ureter, displacement of the kidney, twisting of the ureter, pregnancy, and the like, favors the production of pyelitis.

Morbid Anatomy.—In the milder cases there are swelling and redness of the mucous membrane of the pelvis and a catarrhal exudate. In the severer forms the pelvis is greatly thickened, indurated and filled with pus. The straight tubules of the kidney are involved and show as radiating whitish or yellow streaks in the cortex. Small or large abscesses may be found in the substance of the kidney or the whole organ may be converted into one great sac of pus.

Bacteriology.—In the great majority of cases the colon bacillus is found. In others the pyogenic cocci, or any one of a great variety of bacteria, proteus vulgaris and others.

Symptoms.—In the milder grades there are some slight tenderness over the kidneys and the appearance of some few leukocytes with mucus in the urine. In the severer suppurative forms the patient presents (1) constitutional symptoms of infection, fever, possibly chills and sweating, more or less rapid pulse, anemia and emaciation; (2) local signs, such as pain and tenderness over the kidney with enlargement of the organ and in severe cases rigidity of the overlying abdominal muscles.

The COURSE may be either very acute with severe chills and the clinical picture of sepsis, or very slow with little or no fever, a little pain and tenderness, and gradual loss of vigor. The milder cases recover in a few days or weeks. The severer go on to abscess formation in the kidney and require drainage or removal of the organ.

The BLOOD shows a leukocytosis of moderate or severe grade according to the reaction to the infection. The ratio of polynuclears will be increased, as a rule, in suppurative cases.

Urine.—The urine is decreased, normal, or increased in amount, and the specific gravity varies accordingly. The reaction is acid, neutral or alkaline. Pus is present, in large or small amounts; occasionally it may be absent, if the ureter is blocked. Very rarely fragments of kidney tissue are found.

Diagnosis.—This must be based on the combined constitutional and local signs and the urinalysis. The source of the pus may be inferred from the localization of the pain and the tenderness and possibly tumor of one kidney. Cystoscopy, or, better still, catheterization of the ureters, may show conclusively the side from which the pus comes. The tubercular forms can be differentiated only by the demonstration of the tubercle bacillus in the urine. For conclusive results the organism must be tested by inoculation in animals.

Treatment.—Rest in bed, abundance of water, a nutritious diet, and urotropin are employed in the milder cases. Urotropin is given in doses of 7 to 8 grains three times daily. If these measures do not control the process, the kidney must be incised and drained or removed in toto. Vaccines have been tried, but have little effect.

NEPHROLITHIASIS

(Renal Calculus.   Stone in the Kidney.   Gravel)

Definition.—The deposition of concretions either in the parenchyma or pelvis of the kidney. The majority are composed of uric acid and its allies, of calcium oxalate, or the phosphates of calcium and magnesium. A stone is rarely made up of one ingredient, regularly several substances are found, with one predominating.

Calculi.—Stones composed chiefly of uric acid are hard, angular and reddish in color. Calcium oxalate also produces very hard calculi, usually small in size, gray or blackish in color. The phosphatic stones are likely to be soft and whitish in color. Chemical analysis is necessary to establish the composition accurately.

Etiology.—Calculi are met with at all ages, even at birth. Clinically they are most frequently seen between 30 and 40 years. The tendency to the formation of calculi is transmitted in certain families. The causes of the deposition of urinary constituents must vary in different cases, as the uric acid and urates are precipitated in acid urine, the phosphates in alkaline, and the oxalate of lime in urine, either alkaline, neutral, or acid. Errors of diet, the drinking of too little water, and lack of exercise are usually given as indirect causes. Excessive eating of meat is held to favor the formation of uric acid stones, but there is no agreement as to the dietary irregularities underlying the condition.

Morbid Anatomy.—Stones are single or multiple. They are usually found in the pelvis of the kidney, but may be imbedded in its substance. Not rarely they are found in the ureter, in which there are three points

likely to be the location of a stone: (1) just at the beginning of the
ureter, (2) at the crossing of the iliac vessels, (3) the terminal portion.
The pelvis of the kidney is often dilated, and may show the lesions of
pyelitis. In advanced cases the kidney is involved and shows the con-
dition of pyelonephritis. In the absence of suppurative lesions the
kidney frequently presents an advanced interstitial nephritis.

Symptoms.—(1) There may be none, although several stones are
present in the kidney. Pyelitis, hydronephrosis, or chronic nephritis
may result without symptoms suggestive of stone. (2) Pain more
or less constant and of varying severity may be present in the affected
side. The pain is referred to the region of the diseased kidney, or if
severe may radiate to the other. Nausea, vomiting, or headache, may
be associated. (3) Attacks of renal colic may occur. The onset is
sudden, with violent pain in the affected side, radiating downward into
the scrotum and inner side of the thigh, rarely into the back. Fever
of 102° to 103° F. follows, and nausea or vomiting results if the pain
is severe. The urine is passed frequently, but with difficulty. The
patient is usually prostrated during the pain, which lasts for from
several hours to several days. After one attack the stone may be
passed and no more follow. Or the stone may remain in the bladder
and give rise to characteristic symptoms. If the stone remains in the
pelvis the attacks may be repeated at any time, sometimes frequently,
in other cases only after years. Pyelitis, hydronephrosis, or chronic
interstitial nephritis may result. Following the attack of renal colic,
blood in a trace or considerable amount is usually found in the urine.
The affected side of the abdomen may be sensitive to pressure and the
kidney may feel large.

Diagnosis.—The location and radiation of the pain are suggestive
only. The presence of blood in the urine with the pain is important.
Tenderness over one kidney, if present, is usually reliable. Cystoscopy
is helpful in determining the question of the conditions in the bladder.
The X-ray is invaluable, regularly locating the stone. Ureteral catheter-
ization is necessary to determine the functional power of the other
kidney, if removal of the diseased one is contemplated. The condition
must be carefully differentiated from appendicitis, gall-stone colic,
salpingitis, intestinal colic, mucous colitis, or other affections of the
kidney due to displacement, torsion of the pedicle, or tumor.

Prognosis.—The attacks of pain or colic are not often grave in
themselves. Recurrent attacks may be severe. The development of
pyelitis, hydronephrosis, or chronic nephritis is of serious import.

Treatment.—Renal colic, if severe, is best treated by a hot bath and
an injection of morphine sulphate, 1/6 to 1/4 grain, or the inhalation
of a little chloroform. Hot poultices or fomentations to the side may
be used. To prevent the formation of more calculi, a moderate diet
with abundance of water and reasonable exercise must be prescribed.
If the composition of the stone is known or can be inferred from the
examination of the urine, the diet can be modified to reduce excess in

urates, phosphates, or oxalates.   For uric acid calculi the urine may be kept alkaline, for phosphatic calculi acid; and the possibility that small stones may be dissolved must be admitted.   For large or impacted calculi operation is required.   Calculi lodged in the lower end of the ureter may be dislodged by massage through the rectum.

HYDRONEPHROSIS

Definition.—A distention of the pelvis of the kidney, often associated with atrophy of the substance of the organ, due to obstruction of the ureter and retention of urine.

Etiology.—The causes are sometimes congenital, such as phimosis or stricture of the ureter.   It is usually an accident of adult life, due to obstruction of the ureter by calculi or twists or tumors, or tumors of the bladder, enlarged prostate or stricture of the urethra.

Morbid Anatomy.—The condition is unilateral unless the obstruction is in the urethra.   The pelvis is dilated to a greater or less degree and the kidney shows corresponding atrophy.   In rare cases it is converted into a mere shell.

Symptoms.—There may be localized pain and tenderness, especially if the obstruction is suddenly developed.   In most cases the chief symptom is the tumor.   This is tense, elastic, and possibly fluctuating, if large enough to be definitely palpated.   It is often variable, emptying and filling again from time to time.   It may have the situation of the kidney or occupy any part of the abdomen.   If large, it may cause symptoms by pressure.

Diagnosis.—The tumor must be differentiated from other cystic tumors of the abdomen, ovarian, pancreatic, or mesenteric cysts, and distended gall-bladder.

Prognosis.—Hydronephrosis may exist for years without serious symptoms.   The greatest dangers are rupture into the peritoneum, or suppuration.

Treatment.—The removal of the cause of obstruction by surgical measures is called for.

PERINEPHRITIC ABSCESS

Definition.—A suppurative inflammation of the connective tissue about the kidney.

Etiology.—The disease is usually secondary to suppuration, either in the pelvis or cortex of the kidney.   It may result from the burrowing of an empyema, or an appendix abscess.   It sometimes follows trauma, such as a blow.

Morbid Anatomy.—An abscess cavity is formed which may burrow upward and involve the pleura, or downward along the psoas and present in the groin.   The kidney may or may not be involved.

Symptoms.—The symptoms of the condition are usually very vague until the abscess develops and can be located in the loin.   (1) General symptoms: fever is present, continued, with moderate remissions, or of

distinctly septic type (see Fig. 30). The pulse is a little quickened. The patient feels weak and sick. Sweating and anemia may be marked. (2) Local symptoms: There is pain in the loin or on one side of the abdomen and some rigidity of the abdominal muscles on examination. (3) Sooner or later a swelling appears in the loin, with pain and tenderness. Aspiration or incision reveals the presence of pus. The urine is usually normal, but if a pyelitis is present will show pus.

Diagnosis.—This must be based on the general symptoms of sepsis and the local signs of abscess.
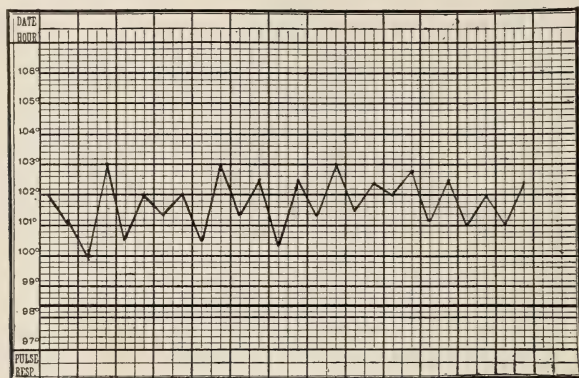
FIG. 30.—Perinephritic abscess with temperature resembling typhoid fever

Prognosis.—This depends upon the cause. The majority recover promptly on the opening of the abscess, but the disease may be fatal.

Treatment.—Incision with careful drainage is necessary.

TUMORS OF THE KIDNEY

Benign tumors of the kidney, such as adenomata, lipomata, fibromata, are found at operation or post-mortem, but are not of clinical importance. Malignant tumors, sarcoma and carcinoma, are not infrequent. Sarcoma of the kidney is seen from time to time in children, but adults are most often affected. Cancer is found only in adult years.

Symptoms.—(1) The kidney involved increases steadily in size and may become rough or nodular. The tumor may press upon or involve surrounding organs. (2) Pain is usually present. (3) Hematuria is a frequent symptom. (4) Cachexia and emaciation develop.

Diagnosis.—Tumors of the kidney require to be differentiated from those of the liver, spleen, and other adjacent viscera. Tumors of the adrenal (hypernephroma) are regularly mistaken for renal growths and can rarely be satisfactorily distinguished except by the study of sections.

Treatment.—Removal is sometimes successful and should be undertaken early.

DISEASES OF THE CIRCULATORY SYSTEM

DISEASES OF THE PERICARDIUM

PERICARDITIS

Definition.—An acute or chronic inflammation of the pericardium. Several clinical forms are described: (1) Acute plastic, fibrinous or dry pericarditis; (2) pericarditis with effusion, serous, purulent, or hemorrhagic, and (3) chronic adhesive pericarditis. In both etiology and pathology there is a close similarity between pleurisy and pericarditis.

1. Acute Fibrinous Pericarditis. 2. Pericarditis with Effusion. The ETIOLOGY of these two forms is practically the same and as pericarditis with effusion is only a further development of the dry or fibrinous form they may well be considered together. The affection develops in rare cases primarily, but in most instances is secondary. It is met with at all ages. (a) Primary or idiopathic pericarditis is a very rare affection. Doubtless most of the apparently primary cases are in reality secondary to some antecedent infection, especially tuberculosis. (b) Secondary pericarditis. (1) Acute rheumatic fever is by all means the most common cause. In children pericarditis may develop in individuals who have shown very few evidences of rheumatic infection; it may follow chorea or the tonsillitis common in rheumatic subjects. (2) Scarlet fever, influenza, or pneumonia may be complicated by pericarditis. (3) Septicemia, pyemia, or any other type of septicemic infection, such as puerperal sepsis, or malignant endocarditis, may cause pericarditis. (4) Chronic interstitial nephritis, gout, or diabetes, may be followed by pericarditis. (5) Tuberculosis is a frequent cause, especially in the generalized type of the disease or in cases of tubercular pleurisy. (6) Any acute inflammatory process in the lungs or pleura (pneumonia) may by direct extension involve the pericardium.

Bacteriology.—Some cases of acute pericarditis are sterile, but in all probability most of the cases are due to bacterial infection. The ordinary pyogenic bacteria, the streptococcus, staphylococcus, pneumococcus or gonococcus, and the tubercle bacilli are most often found.

The streptococcus rheumaticus of Poynton and Payne has also been found in pericardial effusions. The apparently primary cases are likely to yield the tubercle bacillus. The organism is sought by the same methods as are applied to pleural fluids.

Morbid Anatomy.—In the simplest form there is dry fibrinous exudate upon one or both surfaces of the pericardium. The fibrin may be limited to isolated patches or cover the whole surface. (See Fig. 31.) In other cases there is a thick fibrinous exudate coating the heart and

parietal pericardium and giving a very rough, shaggy appearance to the heart. In either case there is a slight exudate of clear or turbid serum in the pericardium. The fluid effusion in some cases becomes so abundant as to constitute the chief feature of the condition, which is then termed pericarditis with effusion. The effusion consists of a clear or turbid serum or, especially in septic cases, of pus. In rare instances blood is present in sufficient amount to tinge the serous or purulent

Fig. 31.—A heart covered by the thick exudate of fibrinous pericarditis. The upper third of the specimen is the reflected pericardium held by the pathologist's fingers.

effusion. In all cases of marked inflammation of the pericardium the heart muscle is affected to some extent and on examination the myocardium shows degenerative or inflammatory changes.

Symptoms.—Acute fibrinous pericarditis frequently gives no distinctive symptoms and is disclosed only by careful examination of the heart. It is often overlooked and discovered only at autopsy. If symptoms are presented they, as a rule, consist of (1) Precordial dis-

comfort or pain, usually slight but in some cases severe. (2) Increased frequency of the pulse and force of the heart action. (3) Slight dyspnea. (4) Fever may be absent, or if present is slight. The temperature curve of the primary affection is unchanged or but slightly raised.

2. Pericarditis with Effusion. Like the dry form of the disease pericarditis with effusion may develop with few or no distinctive symptoms. The amount of the effusion rather than its character seems to determine the severity of the symptoms. (1) Precordial discomfort or pain is usually present and may be intense. (2) Dyspnea becomes increasingly severe with the accumulation of fluid. Orthopnea is often present. (3) Cyanosis, seen especially in the lips and face and under the finger-nails, becomes more and more marked. (4) The pulse becomes rapid, small and irregular, in some cases extremely weak or imperceptible during inspiration, the so-called pulsus paradoxus. (5) The temperature is very irregular. In primary cases there may be an initial chill with subsequent high fever. In the secondary cases the temperature curve usually shows no definite change, or there may be a slight increase. The course of the fever after the establishment of an effusion is very variable. The fever continues, as a rule, as long as there is any effusion in the pericardium.

Physical Signs.—(1) ACUTE FIBRINOUS PERICARDITIS.—Inspection shows nothing significant. Palpation may in marked cases detect a friction rub synchronous with systole of the heart, and analogous to the friction rub sometimes felt over a dry pleurisy. Percussion shows no marked change in the heart area. Auscultation regularly discloses a characteristic dry, to and fro, friction sound or murmur produced by the rubbing together of the inflamed pericardial surfaces as the heart contracts and relaxes. The sound or murmur therefore has the rhythm of the heart action. It usually seems superficial, close to the ear, and is much harsher and dryer than an endocardial murmur. It may be heard over any part of the heart, but most often is detected at the base, and is not transmitted beyond the limits of the heart. Similar sounds may be produced by pleurisy in the part of the pleura overlying the heart. These so-called pleuro-pericardial murmurs are usually easily recognized by the fact that they are greatly modified or obliterated by having the patient stop breathing during auscultation. If produced in the pleura the murmur or rub, as a rule, ceases for a time, to return with renewal of respiration.

(2) PERICARDITIS WITH EFFUSION.—The early signs are commonly those of dry pericarditis. As the fluid accumulates the signs are modified. Inspection: In children a bulging of the precordial area is frequent. In adults the more rigid chest wall does not yield so easily, but the precordial area may become prominent. The respiration is rapid and superficial with slight motion of the thorax, especially on the left. The apex impulse is not visible in the normal site. An impulse may be observed in the third or fourth spaces. The diaphragm and left lobe

of the liver may be depressed and the epigastrium become prominent. Cyanosis may be noted in the lips or face and under the finger-nails. The expression of the face is often anxious and distressed. Palpation: The cardiac impulse is not felt at all or is found in the third or fourth space. The friction rub is lost or can be felt only at the base. Fluctuation cannot be made out through the chest wall. Percussion: The area of cardiac dulness is enlarged both to left and right, and if outlined on the chest has a characteristic triangular form with a much broader base than normal. (See Fig. 32.) The resonance regularly present in the fifth intercostal space to the right of the sternum (the cardio-
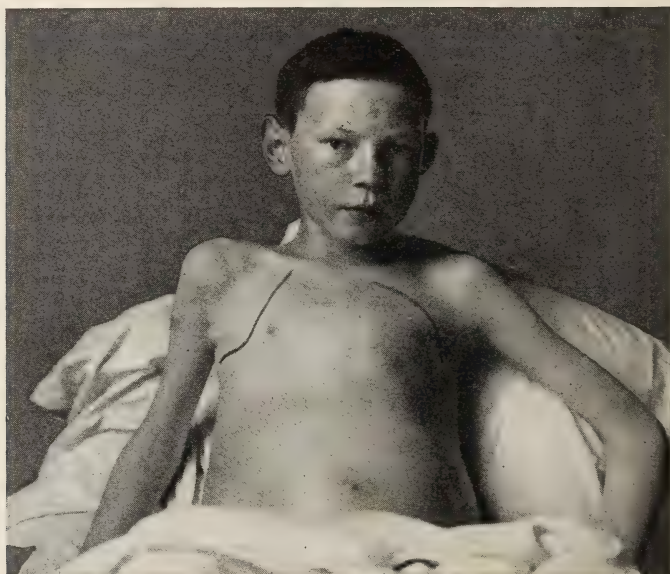
Fig. 32.—The enlarged area of cardiac dulness and the obtuse cardiohepatic angle of pericardial effusion.

hepatic angle) is replaced by dulness. If carefully followed the area of cardiac dulness is found to increase steadily with accumulation of the fluid from day to day, or in severe cases from hour to hour. (See Fig. 33.) In any suspected case the outlines of the cardiac dulness should be carefully marked upon the chest wall for comparison. Auscultation: The characteristic feature is the suppression of the cardiac sounds. The more abundant the fluid the fainter these become, till they can scarcely be heard. If a friction sound has been heard it is lost, or limited to the base, but returns with subsidence of the fluid. Signs over the left lung: In the left infrascapular region with any large effusion, particularly in children, there may be an area of dulness, diminished or bronchovesicular voice, and breathing, and diminished

vocal fremitus, suggestive of pleural effusion or pneumonia. These signs are produced by compression of the left lower lobe. As an effusion may be present in the thorax as well as in the pericardium, the differential diagnosis is extremely difficult, and doubt can be removed only by the careful use of an exploring needle.

Course and Prognosis.—Acute fibrinous pericarditis regularly runs its course in a few days and subsides without serious after-effects. The pericardial layers may be left adherent, but unless this is complicated by a chronic mediastinal and pleural inflammation, no serious results need be expected.

Pericarditis with effusion runs a very variable course, sometimes brief, resolving in a few days, in other cases protracted for weeks or
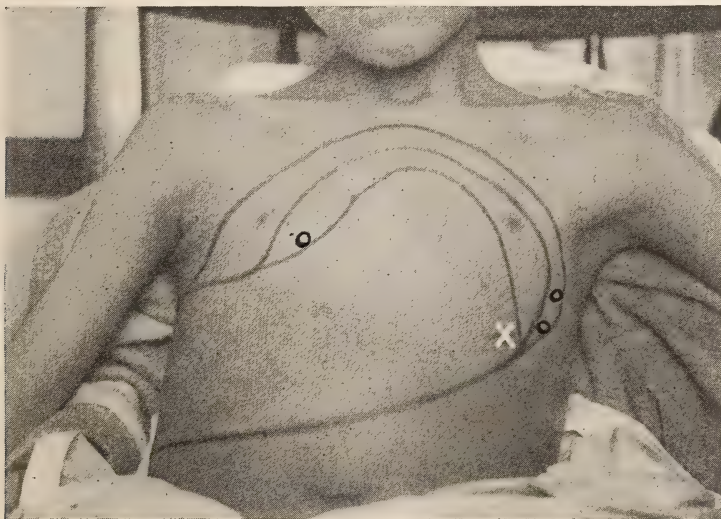
Fig. 33.—Outlines of cardiac dulness at successive stages of pericardial effusion, fusing below into liver dulness. The X marks the apex impulse; the circles the preferable points for aspiration.

months. Rheumatic pericarditis regularly resolves, even when the effusion is abundant, in the course of two or three weeks. As a complication of pulmonary or pleural inflammation pericarditis with effusion adds greatly to the danger of the patient. So long as the effusion remains clear the outlook is hopeful, but if, as often happens, the inflammation becomes suppurative, the prognosis is very grave. In unfavorable cases the temperature rises, the pulse fails, cyanosis and dyspnea become extreme, delirium and coma develop and death quickly follows. In some cases the fatal issue is protracted for weeks and the patient dies of exhaustion.

The Blood.—A leukocytosis is present, as a rule, in all forms except the tuberculous. The degree of leukocytosis varies greatly and very high counts (30,000 to 40,000) may be had in cases with serous effusions,

but a steady increase in successive counts is always suggestive of the development of pus. The differential counts and the cytology of the fluid must be interpreted on the same principles as in the case of pleural effusions. Serous effusions may be tested for tuberculosis by inoculation.

Diagnosis.—The pericardial friction is the one sure sign of acute fibrinous pericarditis. The distinction of this sound from an endo-cardial murmur is not difficult. Pleuropericardial sounds may be distinguished by the influence of respiration upon them.

The diagnosis of pericarditis with effusion also rests in great part upon the physical signs. The enlarged area of cardiac dulness, the dulness in the cardiohepatic angle, faintness of the heart sounds, with symptoms of cardiac and respiratory embarrassment, may be confused with cardiac dilatation. In the latter the cardiac impulse, though faint, is often seen and felt, the sounds are not so faint, endocardial murmurs are usually present and the cardiohepatic angle is preserved. A peri-cardial friction sound may be heard at the base of the heart, even with fluid in the pericardial sac. In cases under observation the surest sign of pericardial effusion is progressive enlargement of the area of cardiac dulness. This can be determined only by carefully outlining the limits of dulness from day to day.

(3) Purulent Pericarditis. This may follow a serous effusion or the exudate may be purulent from the outset. The purulent effusion is most often seen in pneumonia, in septic infections and tuberculosis. In onset, symptoms and course, it can be distinguished from the serous effusion only by the aspirating needle. Various organisms, pneumococcus, streptococcus or staphylococcus, or the tubercle bacillus may be found in the pus. The prognosis is always grave.

(4) Hemorrhagic Pericarditis. Blood may be mingled either with a serous or purulent effusion. Its presence has no peculiar significance and does not affect the course or prognosis of the disease.

Treatment.—Absolute rest is the first requirement. For the relief of pain or distress an occasional dose of codeine or morphine may be required. To quiet the heart's action an ice cap may be applied over the heart. Blisters are now rarely used. The feeding must be carefully regulated and the bowels kept open. For large effusions aspiration may be required, though it is surprising how often patients with serous effusions of considerable amount, especially in the rheumatic cases, recover without intervention. Puncture for diagnosis or aspiration is made either in fifth right interspace in the fourth or fifth spaces to the left, or in the area of cardiac dulness outside the apex, if this can be located. The position of the internal mammary artery is to be re-membered and the needle inserted either close to the sternum or more than an inch from it. Aspiration may be repeated. For purulent effusions free incision and drainage are necessary. Strychnine, digitalis, or other cardiac stimulants may be required to strengthen the heart action.

CHRONIC ADHESIVE PERICARDITIS

(Adherent Pericardium)

Chronic Adhesive Mediastinitis.—Following any acute inflammation the layers of the pericardium may be left adherent, the adhesions being slight or dense. If this be all, no harm results, and in some cases the adhesions seem to disappear in time. A condition in which with the adhesion of the layers of the pericardium there is combined a chronic adhesive inflammation of all the structures in the mediastinum, binding the pleura to the pericardium and the chest wall, and to the diaphragm, and thus causing the heart to be constantly pulled upon by the expansion of the chest in respiration. The heart is regularly found greatly hypertrophied and dilated.

Symptoms.—Simple adhesion of the leaves of the pericardium gives no symptoms or signs and is recognized only at autopsy. Chronic mediastinitis with adhesions of the pericardium gives no symptoms in the early stages, but ultimately causes hypertrophy and dilatation of the heart, and the patients come under observation with evidences of enlarged heart and signs of cardiac failure, such as dyspnea, cyanosis and edema.

Children or young adults, especially those with a history of rheumatism, are most often affected, and the condition is rarely observed among adults. Certain signs are suggestive of it. (1) Retraction of the chest wall over the apex or in the epigastrium with systole. (2) Systolic retraction of the tenth and eleventh intercostal spaces below the angle of the scapula (Broadbent's sign), due to adhesions between the diaphragm and the heart, is a valuable sign. (3) Diastolic collapse of the veins of the neck (Freidreich's sign) may also be observed. (4) With these signs evidences of cardiac failure, dyspnea, cyanosis, rapid and irregular pulse, enlargement of the liver and anasarca are usually associated. (5) Fixity of the apex impulse with the patient lying on his back or side and failure of the left lung to modify markedly the area of cardiac dulness on deep inspiration are also evidences of adhesion. (6) Auscultation usually reveals the presence of murmurs due to insufficiency produced by dilatation, mitral systolic, possibly aortic diastolic and pulmonary systolic. A murmur indistinguishable from that of mitral stenosis has more than once been heard in these cases.

Diagnosis.—This can rarely be made with any degree of certainty, but the condition may be suspected in cases presenting the characteristic signs. Adherent pericardium is often discovered only at autopsy. The condition is usually regarded as endocarditis with subsequent hypertrophy and dilatation of the heart. Prognosis depends largely upon the age and the condition of the heart. In most cases giving clinical evidence of the condition the heart muscle is seriously involved and the prognosis is bad. Some patients, on the other hand, do well for years. The younger the patient, as a rule, the worse the prognosis. TREATMENT is that of the resulting cardiac hypertrophy and dilatation, rest, the use

of digitalis, cathartics, etc., as necessary. Resection of the ribs and cartilages to which the pericardium has become bound has been advocated.

TUBERCULOUS PERICARDITIS

Tuberculosis of the pericardium is, strictly speaking, always secondary to a primary disease in the mediastinal lymph-nodes, the lungs, pleura, or other part. When the primary affection is latent, as it may well be, in the lymph-nodes, the pericarditis is often spoken of as primary. The pericardial affection may be of the dry form or may be accompanied by effusion, serous or more rarely purulent. A bloody serous effusion is particularly suggestive of tuberculosis. The clinical course of tuberculous pericarditis is likely to be sub-acute or chronic in type. The symptoms and signs of the disease are those already described for the dry or serous pericarditis or the chronic adhesive form. The tuberculous nature of the affection cannot be determined on clinical grounds; the diagnosis must rest either on the presence of known tuberculosis in the lungs, pleura, or other part, or on the demonstration of the tuberculous character of the exudate, in case fluid is obtained. This may be accomplished in the same manner as in pleural effusions. (See page 56.)

Cancerous pericarditis is a rare disease, always secondary to cancer elsewhere, particularly of the lungs or pleura. Here also hemorrhagic effusions are common. The nature of the disease can be determined only by a knowledge of the primary growth.

OTHER AFFECTIONS OF THE PERICARDIUM

Hydropericardium.—A non-inflammatory accumulation of serum in the pericardium (a transudate). Etiology.—The condition may be part of a general anasarca, occurring under the same conditions as hydrothorax. (See page 54.) The symptoms and signs are those of pericarditis with effusion, but without fever, pain, or pericardial friction. Increasing dyspnea is usually the prominent feature. The treatment is that of the anasarca, or aspiration.

Chylopericardium is an exceedingly rare condition, like chylous ascites, resulting either from disease of the lymphatic vessels (thoracic duct) or from a fatty degeneration of cells in a pericardial exudate. The signs are those of pericardial effusion. Hemopericardium, distention of the pericardium with blood, results either from penetrating wounds, the rupture of an aneurism of the aorta, or a rupture of the heart wall. The symptoms are those of hemorrhage with rapid distention of the pericardium. The condition is usually fatal within a few hours. A few cases of successful operation for wounds of the heart wall with hemopericardium are on record.

Pneumopericardium and hydropneumopericardium or pyopneumopericardium are analogous to similar conditions of the pleura. They

result from penetrating wounds or the rupture of ulcer, cancer or tuberculosis, of the stomach, esophagus, or lung into the pericardium. Gas may be formed in the pericardium by the action of such organisms as the bacillus aërogenes capsulatus. An active pericarditis regularly accompanies the condition. The symptoms are those of acute pericarditis with effusion. The signs are those of shifting dulness with tympany over the higher portion of the pericardium and remarkable churning and splashing sounds with feeble, distant or inaudible heart sounds. The prognosis is naturally very grave. The treatment consists of incision and drainage of the sac.

DISEASES OF THE HEART

ENDOCARDITIS

Definition.—An inflammation of the endocardium, commonly confined to the valves of the heart.

We describe an acute endocarditis, either simple or malignant, and chronic endocarditis. For convenience endocarditis and myocarditis are always described separately, but clinically they are very frequently associated.

ACUTE ENDOCARDITIS

Etiology.—Children are most often affected, but the disease is met with at all ages. Two factors are of importance. (1) Infection. Probably all cases of acute endocarditis are due to blood infections of some kind. Any condition in which micro-organisms are present in the blood may give rise to an acute endocarditis. An acute infectious disease is therefore the usual antecedent. Certain acute infections—especially those due to cocci—are much more likely to cause endocarditis than others. Rheumatic fever causes at least 50 per cent. of all cases, then follow pneumonia, septicopyemia, scarlet fever and gonorrheal infection. Bacilli, whether typhoid, tubercle, or diphtheritic, though present in the blood, rarely cause endocarditis.

(2) Conditions favoring the lodgment of the organisms on the heart valves. Valves damaged by preceding endocarditis are especially vulnerable, so that chronic endocarditis is likely to be complicated by repeated new infections. Valves damaged by trauma or subjected to unusual strain are also susceptible. Of the other influences determining the lodgment of bacteria on the heart valves, we know little.

The portal of entry of the bacteria can most often not be determined. In rheumatic fever, and possibly other infections, the tonsils are accused. Rarely an infected wound of the surface of the body is followed by endocarditis. Uterine infections are most frequently the avenue of entrance to the blood and heart.

Morbid Anatomy.—VEGETATIONS on the heart valves, rarely on the heart wall, are the characteristic lesions. The vegetations are commonly small, but may be large, wart-like growths. They are composed in the

early stages of fibrin, leukocytes and often bacteria, later there is a growth of connective tissue and a proliferation of the endothelium of the heart valve. The mitral and aortic valves are most often affected, rarely those of the right side.

In the severer cases the formation of vegetations is associated with ULCERATION (see Fig. 38), resulting in the destruction of more or less of the heart valves. When the heart wall is affected, it may be softened or even perforated. With the ulcerative process the vegetations are regularly larger and more abundant.

With either large or small vegetations, with or without ulceration, vegetations may become loosened from the heart valves and lodge in any part of the body, the brain, spleen, kidneys, lungs, etc., and cause the usual evidences of embolism. These occurrences are naturally more common in severe cases, marked by ulceration and the formation of large vegetations.

As acute endocarditis subsides the vegetations become organized by conversion into or replacement by connective tissue, and the valves may be deformed by contraction, as in chronic endocarditis.

Bacteriology.—The cocci are most often found, especially streptococci. Staphylococci and pneumococci, rarely gonococci, are occasionally observed. Bacilli, such as the typhoid, colon, diphtheria and tubercle bacilli, are very rarely found.

While the distinction between simple and malignant endocarditis is one wholly of degree, and no sharp line can be drawn between them, it serves a useful purpose clinically and is commonly observed.

SIMPLE ACUTE ENDOCARDITIS

This includes the type of the disease occurring in rheumatic fever and most of the acute infectious diseases. The lesions are mild, the symptoms are usually slight, and bacteria cannot, as a rule, be found in the blood.

Symptoms.—These are often entirely lacking or lost in those of the primary disease. There may be slight fever, increase of the pulse rate, possibly with irregularity, and discomfort or actual pain about the heart.

Physical Signs.—The significant sign is the development of a murmur over the mitral or aortic area which is persistent and possibly increases with time. The heart may be dilated, its action over-forcible or irregular, with a corresponding pulse. Embolic phenomena are very rarely met with.

Course.—The symptoms of simple endocarditis usually subside in the course of a few weeks. The affected valve or valves return to normal, or they are left permanently damaged and go on to present evidences of chronic endocarditis.

Treatment.—Rest is the chief measure. For rapid or excited heart action an ice-cap may be applied over the heart. In rheumatic cases the salicylates should be given in full doses. The patient must be kept

quiet and nursed with the utmost care till the symptoms and signs disappear or until the lesion present is thoroughly compensated. This may require rest in bed for several months. The diet must be carefully regulated to the digestive capacity, and the tendency to anemia met by fresh air and iron. Return to active life must be made gradually.

MALIGNANT ENDOCARDITIS. SEPTICEMIC ENDOCARDITIS

Malignant endocarditis differs only in degree from the simple form. The difference lies chiefly in (1) that in malignant endocarditis organisms are regularly found in the blood. From this standpoint the disease is therefore a septicopyemia with lodgment of the organisms on the heart valves. (2) The formation of vegetations and destruction of the heart valves is more pronounced than in the simple form and the damage done to the heart is therefore regularly greater. The organisms found in the blood are strepto-, staphylo- or pneumococci, rarely gonococci, and still more rarely bacilli, such as the typhoid, diphtheria or tubercle bacilli. The streptococcus mitior seu viridans is commonly found.

Etiology.—The disease may complicate any form of septicemia, particularly the septic infections of the uterus, infected wounds of any kind, pneumonia, or gonorrheal septicemia. In most of the cases no source for the septic infection can be found.

The endocarditis of rheumatic fever rarely assumes the malignant type, and the disease is therefore rare in children. Old endocardial lesions favor the lodgment of bacteria upon the valves, and doubtless for this reason malignant endocarditis is most often seen in early or middle adult life in persons having had rheumatic endocarditis.

Morbid Anatomy.—The vegetations in malignant endocarditis are usually large and numerous and the destruction of the heart valves and possibly of the heart wall marked. Emboli are numerous in the brain, lungs, liver, spleen, kidneys, or possibly in the large vessels of the extremities. They appear first as reddish areas of infarction, later becoming paler as the blood is absorbed and connective tissue develops or necrosis takes place.

Although pyogenic bacteria are present in these emboli the infarcted areas rarely suppurate. Septic meningitis may, however, be caused or abscesses in the viscera, the joints, or other parts of the body. The characteristic petechial rash may be present on the skin. Anemia is often profound.

Symptoms.—These vary greatly in different cases, the variations depending chiefly upon the prominence of the symptoms of septic infection and the damage done to the heart. The evidences of sepsis are sometimes intense and the patients die before much damage has been done to the heart. In other cases the septic phenomena are very slight. In like manner the evidence of damage to the heart may be either very marked or altogether lacking.

Several clinical types may be described.

1. The typhoid type in which the fever, headache, delirium, rapid pulse and profound prostration are the marked features. Abdominal distention, enlargement of the spleen and diarrhea may also be present and the resemblance to typhoid be close. (See Fig. 34.)

2. The cardiac type in which with the fever and other septic symptoms the heart action is rapid, irregular or intermittent, possibly tumultuous; there are loud, harsh murmurs, pain about the heart, and possibly dilatation, to direct attention to that organ.

3. The meningeal type, in which headache, delirium, coma, or convulsions, rigidity of the neck and general hyperesthesia suggest meningeal involvement. The eruption of malignant endocarditis is often not to be distinguished from that of cerebrospinal meningitis.
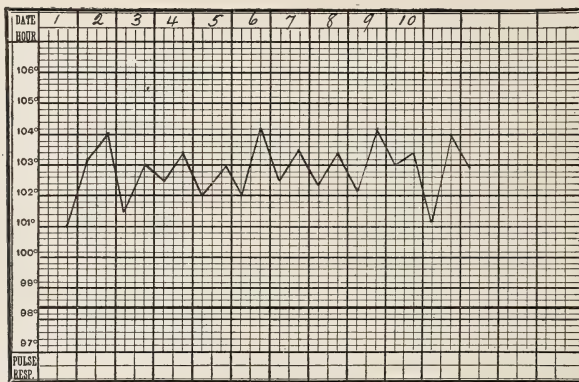
FIG. 34.—Malignant (septicemic) endocarditis fever curve resembling typhoid fever.

4. The chronic septic type, in which the chief feature is a septic fever with sweating and progressive anemia lasting for months or even a year. Leukocytosis may be lacking in these cases.

For the diagnosis of malignant endocarditis we require, as a rule, (1) Fever, either continuous, or of the remittent or intermittent septic type. Leukocytosis may or may not accompany the fever.

(2) Evidences of endocarditis. These are usually loud, harsh murmurs, most often over the mitral, sometimes over the aortic valves, usually murmurs of insufficiency, rarely of stenosis. Dilatation and hypertrophy may or may not be present.

(3) The demonstration of micro-organisms in cultures from the blood.

(4) The characteristic rash or other embolic phenomena. The rash consists of minute red petechial spots, occurring on any part of the trunk or extremities, especially on the forearms or the legs. Occasionally they are seen in the conjunctivæ or on the mucous membranes of the mouth and throat. This rash is produced by the lodgment of minute emboli in the skin.

Emboli lodging in the brain give rise to partial or complete hemiplegia, paralysis of ocular muscles, etc. In the lung they cause infarction with pain, cough, expectoration, possibly bloody, and small areas of consolidation.

In the spleen they cause swelling, pain and possibly a friction rub from perisplenitis.

In the kidneys emboli give rise to complaints of pain and possibly hematuria. In the extremities stoppage of the circulation, pallor, coldness and loss of power are the results. Gangrene may result.

In characteristic cases the diagnosis may be made on clinical grounds alone, but the presence of the micro-organisms in the blood is usually necessary to establish it. The course of the disease varies greatly. Most cases are quickly fatal. In the more chronic type life may be prolonged for a year or more. Death is nearly always the outcome. A few cases of recovery under treatment have been recorded.

Diagnosis.—From typhoid fever acute cases may be distinguished by leukocytosis (20,000 or more), the petechial rash, embolic symptoms, and the heart murmurs, while the Widal reaction is negative and blood cultures yield cocci rather than the typhoid bacillus.

From meningitis the results of lumbar puncture and the blood culture usually make the distinction easy. From septicopyemia the evidence of heart lesions, the characteristic rash or other embolic symptoms distinguish it. In the chronic type the disease may be confused with tuberculosis. Pulmonary signs are usually lacking, there is no sputum, or, if present, it does not contain tubercle bacilli, the cardiac signs are present and the rash or other embolic symptoms appear sooner or later. Malaria may be excluded by the absence of plasmodia and the failure of quinine.

Treatment.—Rest, food, fresh air, are employed as for sepsis of any type. In the acute cases morphine may be used for the patient's comfort, there being practically no hope of recovery. In the more chronic type success has been attained in a few cases by the use of autogenous vaccines—that is, vaccines made from the organism obtained from the patient's blood after Wright's method. Streptococcus sera have also been used in appropriate cases, but with no decisive results. Otherwise treatment is purely symptomatic. Transfusion has been resorted to in cases of profound anemia, and has given rise to temporary improvement.

CHRONIC ENDOCARDITIS

Etiology.—There are three chief groups of cases:

(1) Those following acute infectious diseases, especially rheumatic fever. At least 50 per cent. of all cases give a history of rheumatic infection or chorea. In some instances the development of acute endocarditis during the primary disease is known. In most cases the history of rheumatism, scarlet fever, pneumonia, typhoid or other infection is the only clue to the cause of the endocarditis. Owing to the frequency of acute infectious disease in childhood, the chronic endocarditis of those under thirty years of age is usually of this origin.

(2) Those associated with arteriosclerosis and chronic nephritis and due to like causes, especially alcohol, lead, syphilis, or gout. Here also age and hard labor are important factors. The endocarditis of this type belongs to adult life, and especially to the later decades.

(3) In many cases it is impossible to ascertain definitely the cause of chronic endocarditis.

Morbid Anatomy.—The valves of the left heart are regularly affected, those of the right side rarely. The affected valves are thickened, opaque, shrunken, sometimes the various cusps or leaflets are adherent and variously distorted. In the auriculo-ventricular valves the changes in the valve leaflets are accompanied by thickening and retraction of the chordæ tendineæ, which greatly augment the effect of the changes in the segments of the valves. In the late stages of the chronic endocarditis the valves may become extensively calcified.

Vegetations such as appear in acute endocarditis may be found at any stage of the more chronic process and ulceration at times occurs, especially in advanced cases.

As a consequence of chronic endocarditis the chambers of the heart are regularly dilated to a greater or less extent and the walls thinned or thickened. Atheromatous changes in the cardiac arteries are commonly present, especially in the chronic endocarditis associated with arteriosclerosis, together with more or less pronounced changes in the myocardium (chronic myocarditis). Chronic nephritis, usually of the interstitial type, is also commonly associated.

EFFECTS OF CHRONIC ENDOCARDITIS. CHRONIC VALVULAR HEART DISEASE

The Valves of the Heart.—These are rendered incompetent or insufficient so that they no longer properly close the orifices they guard or they are narrowed or stenosed so that they obstruct the flow of the blood through them. Frequently both results are produced. The further effects of insufficiency or stenosis of any valve of the heart vary with the valve affected, the degree of the defect, insufficiency or stenosis, and the secondary changes produced in the chambers of the heart. The heart is a living pump whose satisfactory operation depends not only on the integrity of its valves and chambers, but upon the maintenance of a normal circulation of blood in its walls and also upon normal innervation. The effects of valvular lesions are therefore not adequately stated in purely mechanical terms and yet a clearer understanding of them may be had by tracing out the physiological results of valvular defects. This may best be done with the aid of the accompanying diagrammatic representation of the relations of the cardiac chambers and valves. (See Fig. 35.)

Aortic Insufficiency.—When the aortic cusps are rendered insufficient, a part of the blood thrown into the aorta by the systole of the ventricle is allowed, during the diastole, to flow back into the left

ventricle, while at the same time this ventricle is receiving its usual charge from the auricle. The result is in the first place to overfill the ventricle and so dilate it and secondly by increasing the amount of blood to be thrown out, that is, increasing the work, to cause hypertrophy. These two changes go hand in hand and in the case of aortic insufficiency the dilatation and hypertrophy of the left ventricle are marked and often extreme, producing the so-called cor bovinum.

Dilatation of the left ventricle, when marked, enlarges the left auriculo-ventricular ring to such an extent that the mitral valve can no longer properly close it and a relative mitral insufficiency results, though the valve may be perfect. Aortic insufficiency is therefore regularly,
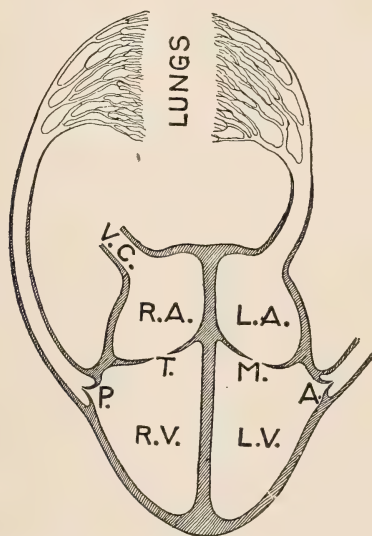
Fig. 35.—A diagrammatic representation of the heart. *V C*, the vena cava; *R A*, right auricle; *R V*, right ventricle; *P*, pulmonary valves; *L A*, left auricle; *L V*, left ventricle; *A*, aortic valve.

in its advanced stages, complicated by mitral regurgitation, and from one point of view a mitral insufficiency may prove advantageous to the patient with aortic insufficiency by lessening the work to be done in emptying the ventricle at each systole. Once mitral insufficiency is produced the heart is subject to the changes incidental to this lesion, just as much as though the insufficiency had been produced by disease of the valve itself.

Mitral Insufficiency.—Incompetence of the mitral valve allows the left ventricle in systole to empty itself not only into the aorta but into the auricle as well, and thus temporarily at least lessens the work of the ventricle. But the left auricle thus unnaturally filled both from the left ventricle and from the right through the pulmonary vessels, systole of the two ventricles being synchronous, must first dilate and

hypertrophy. By such changes the circulation is for a greater or less time maintained practically normal.

Sooner or later, however, the efficiency of the auricle fails, the blood collects in the lungs, the pressure in the pulmonary artery rises, dilatation and hypertrophy of the right ventricle follow with possible incompetence of the tricuspid valve, and ultimately dilatation and hypertrophy of the right auricle.

Aortic Stenosis.—The difficulty in stenosis of the aortic valve lies in the increased force required to empty the ventricle through the narrowed orifice, and consequent imperfect emptying of the ventricle. Hypertrophy and dilatation of the ventricle follow as in aortic insufficiency, but they are less marked and never reach the extreme limits seen in the latter condition. Dilatation of the auriculo-ventricular ring may produce mitral insufficiency and all the chain of events already described.

Because of their close relation to the cusps of the aortic valves the coronary arteries are very likely to be involved in cases of aortic disease, and narrowing or obliteration of the smaller branches results in fibrous changes in the myocardium.

Mitral Stenosis.—This lesion directly increases the work demanded of the left auricle, with resulting dilatation and hypertrophy and an early development of pulmonary congestion, hypertrophy and dilatation of the right ventricle, and incompetency of the tricuspid which must naturally follow. The left ventricle, on the other hand, remains of normal size. The hypertrophy of the heart in mitral stenosis is demonstrable, as a rule, only on the right side. The auricular enlargement, because of the relation of the left auricle to the chest wall, is rarely shown by physical examination.

Primary right-sided lesions such as pulmonary stenosis or tricuspid insufficiency cause dilatation and hypertrophy of the right chambers in a manner analogous to that in which these changes develop secondarily to the lesions of the left side.

COMPENSATION.—When in any cardiac lesion the resulting changes in the heart, hypertrophy and dilatation, permit the circulation to be carried on approximately normally, the lesion is said to be compensated and the process is spoken of as compensation. Eventually in most cases of chronic endocarditis, either by reason of increasing dilatation, or because of changes in the heart muscle or the coronary arteries, the heart becomes incapable of maintaining a normal circulation, and symptoms of cardiac distress and failure of the circulation follow. We then say that compensation is broken.

COMPENSATION AND FAILURE OF COMPENSATION.—In the stage of compensation we may say there are no symptoms, but this statement must always be made with certain reservations. The heart with damaged valves whose defect is relatively balanced by dilatation and hypertrophy of the chambers will often do its work perfectly, so long as the patient leads an inactive life, but in most cases any unusual exertion, especially running or climbing stairs, will cause dyspnea, palpi-

tation and possibly cyanosis, more readily than in the normal individual. So long as these symptoms of distress occur only under unusual conditions they are ordinarily disregarded. When they appear under the ordinary conditions of life, they are regarded as signs of failing compensation.

It is possible that a heart with damaged valves be so adjusted by natural changes that it will do its work even under the severest trial as well as a normal heart. In an instance within the writer's knowledge a student with marked signs of mitral insufficiency, after being warned that he should not enter the contest on account of his cardiac condition, won a mile race against a number of rivals and with less distress than any of them. Such instances of perfect compensation are, however, very exceptional.

Failure, or breaking of compensation, may be caused in several ways: (1) By advancing changes in the heart valves, increasing steadily the inefficient action of the heart; (2) by any influence which increases blood pressure, especially sudden or severe physical exertion, progressive arteriosclerosis, chronic Bright's disease, and the like; (3) by weakening of the cardiac muscle, (a) chronic progressive changes in the cardiac muscle are found in most cases of chronic endocarditis. Such myocarditis gradually impairs the efficiency of the heart muscle, and often in the end determines the failure of compensation. Myocarditis is most marked in those cases of endocarditis in which arteriosclerosis of the coronary arteries is regularly found, *i.e.*, in endocarditis involving the aortic valves. (b) Apart from definite myocardial changes the vigor of the heart muscle may be impaired as that of any part of the body by many influences, especially impaired nutrition from any cause, anemia, acute disease, such as pneumonia or typhoid fever, and the like. Failure of compensation is therefore often a direct result of intercurrent acute disease.

In any of these ways the compensated heart may be rendered unable to longer perform its functions satisfactorily and symptoms develop. The symptoms of broken compensation depend upon the over-strain of the heart and the circulatory changes induced by venous congestion. By reason of the failing power of the left heart the blood pressure falls in the arteries, the difference of pressure between arteries and veins is less than normal, and the blood therefore tends to accumulate in the veins. If the right heart is also affected, the aspirating action of the right chambers on the great veins is lessened and the tendency to venous stagnation is further augmented.

The venous congestion of cardiac failure usually shows itself first in the most dependent portions of the body, the feet and legs, later in the abdomen and the abdominal viscera, especially in the parts drained by the portal vein, then in the lungs and pleura, and finally in the hands, arms and even in the face and neck.

As the venous congestion increases in any part there is a tendency to the exudation of serum from the blood-vessels, with resulting edema.

In the peritoneal, pleural or pericardial sacs such edema is regularly accompanied by an accumulation of free fluid constituting ascites, hydrothorax, or hydropericardium.

Chronic congestion of the mucous membranes causes a chronic catarrhal inflammation which is attended with characteristic changes in structure and function. In the lungs a chronic bronchitis is produced. In the alimentary tract chronic gastritis and chronic enterocolitis.

Chronic congestion of the liver, spleen and kidneys results in enlargement with chronic interstitial inflammation and consequent fibrosis.

In the lungs a similar condition of chronic interstitial inflammation, commonly called brown induration, often results.

SYMPTOMS OF CHRONIC ENDOCARDITIS.—When compensation fails in chronic endocarditis, symptoms develop either suddenly or gradually, depending upon the causes initiating the failure. Whatever the lesion the symptoms of failure are in general produced by progressive venous stasis in the lungs, liver, abdomen and extremities, and the clinical picture is modified but little by the particular valvular lesion. It seems best, therefore, to first describe the common picture of cardiac failure and later indicate the modifications produced by particular lesions.

(1) The heart itself reacts to its increasing burden by producing more or less discomfort or actual pain, by more rapid action, either feebler or more forcible, and by palpitation. In the later stages the action of the heart becomes irregular and intermittent.

(2) Pulmonary congestion declares itself by increasing dyspnea, cough, expectoration and cyanosis. At first the dyspnea is excited only by exertion, especially running or climbing, later it is present even at rest. Often the patient is forced to sit up on account of the dyspnea (orthopnea). Not infrequently the dyspnea is paroxysmal, "cardiac asthma."

The early cough is slight, and attended only by mucous expectoration. Constant coughing and blood-tinged or bloody expectoration mark the severer conditions. These symptoms are often aggravated by the development of hydrothorax. In the final stages pulmonary edema may occur, with profuse watery exudation into the trachea and bronchi, producing intense dyspnea and cyanosis, with characteristic physical signs. Pulmonary edema is the common precursor of death. The sputum in pulmonary congestion often shows blood cells or epithelial cells containing blood pigment.

Cyanosis first shows itself only after exertion under the fingernails or in the lips, later it appears in the skin and is seen even at rest.

(3) The gastric and intestinal congestion produce loss of appetite, nausea or vomiting and constipation, rarely diarrhea. These symptoms vary with the severity of the condition. If vomiting occurs, the vomitus consists of food, or food and mucus. Traces of blood may be seen, but hematemesis is rare. The abdomen may be distended and all these symptoms augmented by the presence of ascites.

(4) The urine becomes scanty, high-colored, and of high specific gravity, with a trace of albumin and hyaline or granular casts. Blood-

cells may be found at any time or a pronounced hematuria may be caused by emboli.

In the extremities edema shows itself, at first appearing only after standing, later becoming constant and extending to the legs, the trunk, hands, arms, and even the face.

I. MITRAL INSUFFICIENCY

Insufficiency of the mitral valve is the most frequent result of chronic endocarditis. It is met with at all ages, but especially in children. It develops in the course of chronic endocarditis, either from thickening and shrinking of the leaflets of the valve or from retraction caused by shortening of the chordæ tendineæ.

Symptoms.—Mitral insufficiency is one of the most easily compensated lesions, often existing for many years without symptoms. Compensation, once thoroughly established, may be protracted indefinitely.

MECHANICS.—When the mitral valve is insufficient, a part of the contents of the left ventricle will, on systole, be forced back through the leaking valve into the left auricle. This chamber must therefore dilate and, in the effort to drive forward an increased charge, hypertrophy. By reason of these changes, it will gradually become capable of driving into the ventricle a larger quantity of blood with each systole. The ventricle will, therefore, gradually dilate and hypertrophy.

The filling of the auricle in diastole not only from the pulmonary veins but also in part from the ventricle raises the tension in the auricle and gradually in the pulmonary vessels. The right ventricle therefore dilates and hypertrophies and the series of changes on the right side already described (see p. 181) is initiated.

PHYSICAL SIGNS.—Inspection.—The precordial region may be prominent or bulge by reason of the hypertrophy of the left heart, especially in children. The apex impulse is displaced downward and outward to a moderate degree. Palpation.—The impulse is forcible, or in extreme cases heaving in character. A systolic thrill is said by some to occasionally accompany mitral insufficiency. There is doubt of this. It is difficult to exactly time a thrill at the apex, and also a certain degree of roughening or narrowing of the valve (stenosis) often accompanies insufficiency. A thrill, presystolic in time, is characteristic of mitral stenosis. Percussion.—The area of deep cardiac dulness is enlarged to the left, usually to a moderate degree. Auscultation.—At the apex a loud, blowing, systolic murmur is heard, either replacing or accompanying the first sound. The murmur is regularly transmitted in all directions to some extent, but especially to the left, and may easily be heard in the back.

The second sound over the pulmonic valve is accentuated, owing to the increased tension in the pulmonary artery. In the later stages there may be evidences of hypertrophy of the right ventricle, pulsation in the epigastrium and increase of cardiac dulness to the right of the sternum, or even of tricuspid regurgitation.

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The pulse is usually regular and of moderate volume but low tension. Only in the late stages does it become irregular or intermittent.

PROGNOSIS.—Mitral insufficiency is probably better borne than any other valvular lesion. In well-compensated cases it may have little influence upon the duration of life. In children, however, any valvular defect is of serious import, because of the difficulty of preventing overexertion in play or sports and consequent disturbances of compensation.

DIAGNOSIS.—There are two conditions likely to mislead: (1) Accidental or hemic murmurs systolic in time and softly blowing in character, heard at the apex and possibly transmitted to some distance from the apex. Such murmurs are met with in the various fevers and anemias and disappear with recovery from the fever or anemia. With such murmurs there is no hypertrophy of the ventricle, no accentuation of the second pulmonic sound, and the murmur itself is softer in quality than those produced by organic lesions. (2) Mitral insufficiency produced by dilatation of the left ventricle, the so-called relative incompetence of the mitral valve. All the signs of mitral insufficiency are found in this case, and only the knowledge of the sequence of events leading up to the development of the murmur or the presence of a presystolic murmur indicative of an accompanying stenosis of the valve will enable one to definitely decide the question. Thus if the murmur of mitral insufficiency develops secondarily to an aortic lesion, either insufficiency or stenosis, or to the increasing strain of high arterial tension from any cause, it can safely be set down as due to dilatation. If, on the other hand, the murmur of mitral insufficiency is accompanied by that of stenosis, it is surely due to disease of the mitral valve itself.

II. MITRAL STENOSIS

This lesion, in a pure form, is a less frequent type of chronic endocarditis. A certain degree of stenosis often develops in the course of mitral insufficiency and the two conditions are frequently associated.

Mitral stenosis is most often met with in women in early adult life. It is not often found in childhood or at advanced age. It is one of the most serious of the valvular lesions.

MECHANICS.—Narrowing of the mitral valve dams back the blood in auricular systole and opposes the normal onward flow. The left auricle therefore hypertrophies and emptying itself incompletely dilates; pressure rises in the pulmonary circuit, and dilatation and hypertrophy follow on the right side. The left ventricle remains of normal size, and often appears contracted in comparison with the enlarged other chambers.

The SYMPTOMS are those already described, but in mitral stenosis signs of pulmonary congestion, dyspnea, cough, and cyanosis are likely to appear early and the pulse is frequently very small and irregular or intermittent.

PHYSICAL SIGNS.—Inspection.—The precordium is usually normal. The apex impulse is not displaced, unless by reason of hypertrophy on

the right side. Cyanosis of the finger-tips or the lips is early noticeable. There may be prominence of the lower sternum and an epigastric pulsation from hypertrophy of the right ventricle. Palpation confirms the findings of inspection. The apex impulse is not forcible. The heart action is often feeble, irregular or intermittent. A short, rumbling, presystolic thrill is regularly felt at the apex, ending in a sharp shock as the ventricular systole begins. Percussion shows little or no increase in the cardiac dulness to the left, a definite increase to the right, as a rule. The pulse is small, and often irregular and intermittent.

Auscultation.—A short, rough, rumbling presystolic murmur closely limited to the apex is characteristic of mitral stenosis. The murmur increases in intensity and ends in a short, sharp first sound or merges into the blowing systolic murmur of a complicating insufficiency.

The murmur is, of course, due to the passage of blood through the narrowed orifice under the impulse of auricular systole. When compensation fails and the auricle dilates, the murmur may disappear, to return with recovery of auricular power. In the absence of the characteristic murmur, the sharp first sound and the accompanying shock, together with the other signs, may enable one to make the correct diagnosis.

Diagnosis.—In its typical form mitral stenosis is easily recognized, yet it is well known that this lesion is very frequently overlooked.

The characteristic murmur may be absent as indicated above, but the presence of the other signs should lead to a correct diagnosis.

Two other conditions give rise to murmurs not easily distinguished from that of mitral stenosis. (1) The so-called Flint murmur, accompanying aortic insufficiency. This is a diastolic or presystolic rumble heard at the apex and attributed to the impact of the regurgitated blood upon the large anterior segment of the mitral valve, causing it to flap and possibly interfere with the free entrance of blood from the auricle. The association of this murmur with that of aortic insufficiency should put the observer on his guard. It occurs in many cases of aortic insufficiency, while true mitral stenosis is very rarely indeed combined with that lesion. The sharp apical first sound and the accentuated second pulmonic are not heard in aortic insufficiency.

(2) In adherent pericardium a murmur indistinguishable from that of mitral stenosis has more than once been heard and has led to error in diagnosis. The reasons for this have not yet been given.

Prognosis.—Mitral stenosis is a grave lesion, but may be well borne for many years. When signs of failure once develop, restoration is difficult, because the strain falls upon the thin-walled auricle and the relatively weak right ventricle. Patients with this lesion rarely reach old age.

III. AORTIC INSUFFICIENCY

Aortic insufficiency ranks next to mitral insufficiency in frequency. It is most often found in men, especially in middle life. It may be produced in different ways: (1) Most frequently it results from an

endocarditis, rheumatic in origin, involving the aortic cusps, thickening and contracting them and uniting their edges, or actually destroying portions of the valves.

(2) It may be part of a general arteriosclerosis affecting particularly the arch of the aorta and the adjacent valves. In these cases extensive calcareous degeneration of the aorta and valves is frequently found.

(3) Segments of the valve may be ruptured by extreme physical exertion, such as lifting heavy weights, with resulting insufficiency. This lesion is therefore common in men engaged in laborious occupations. In animals similar conditions can readily be produced by cutting or tearing one cusp of the valve.

(4) A relative incompetency of the semilunar valves may be produced by dilatation of the aorta due to arteriosclerosis and high arterial pressure, especially in cases in which there is an aneurism of the ascending aorta.

MECHANICS.—In aortic insufficiency part of the blood thrown into the aorta by systole of the left ventricle is allowed by the imperfect closure of the semilunar valves to flow back into the ventricle in diastole. The ventricle therefore fills from both sides, the auricle and the aorta. It must therefore promptly dilate and, having to move an unusual charge of blood, hypertrophy. Extreme grades of hypertrophy and dilatation of the left ventricle are therefore seen more often in aortic insufficiency than in any other condition. The large amount of blood thrown with each systole of the enlarged ventricle into the aorta, and the rapid disappearance of the wave, partly forward into the distal arteries and partly backward into the ventricle, give rise to unusual oscillations in the contents and pressure in the arteries.

Symptoms.—By reason of the relations between the aortic valves and the cerebral arteries the oscillations in the blood stream affect particularly the circulation in the brain. Complaints of headache, dizziness, flashes of light, or faintness upon rising suddenly are rather characteristic of the lesion. The coronary arteries rising from the aorta immediately behind the cusps of the aortic valve are likely to be involved in any process affecting the valves, and patients with aortic insufficiency are therefore more liable to suffer severe cardiac pain or to die of angina pectoris than are those with other forms of valvular disease.

Aortic incompetency may long be well compensated by change in the left ventricle. The circulation is therefore well maintained and these patients often show no traces of cyanosis till late in their disease the ventricle begins to fail and stasis occurs in the pulmonary and peripheral circulation.

PHYSICAL SIGNS.—Inspection.—The precordium is prominent or bulges. The apex impulse is displaced outward and downward, in some cases to an extreme degree, aortic insufficiency producing the largest hearts (*cor bovinum*) one sees. The impulse is often widely diffused and heaving in character. The unusual pulsation of the arteries, especially in the neck, is easily observed. If the patient's hand be raised and

gentle pressure made upon the edge of a finger-nail the red margin will be seen to advance rhythmically with the beat of the heart upon the whitened area (capillary pulse). Similar phenomena may be observed in the uvula or even in the skin of the forehead, if the latter be rubbed to cause flushing. Remarkable pulsation in the retinal arteries may also be observed by the ophthalmoscope.

Palpation.—The displaced apex and its forcible impulse may be felt. The unusual pulsation of the arteries is also noted and not infrequently a thrill is to be felt over the larger arteries, such as the femoral, brachial, radial, or those of the neck. A diastolic thrill may be felt in the aortic area.

Percussion.—The area of deep cardiac dulness is enlarged greatly to the left and downward, corresponding to the position of the apex. The right side of the heart is not affected till late in the disease. The transverse area of cardiac dulness is greater in aortic insufficiency than in any other lesion.

Auscultation.—The characteristic murmur of aortic insufficiency is diastolic in time, heard best in the second intercostal space just to the right of the sternum and transmitted downward under the sternum and along its left margin, even to the ensiform cartilage. Often the murmur is not loudest at the expected site, but at some other point along the course described. It may also be transmitted upward to the clavicle. Over the arteries susceptible of auscultation both systolic and diastolic murmurs may often be heard. The second aortic sound is regularly replaced by the murmur; the second pulmonic is heard as usual or may be accentuated. Since roughening of the aorta often accompanies aortic insufficiency, a systolic murmur may be heard at the base and upward over the great vessels, apart from stenosis of the valve itself.

The pulse of aortic insufficiency is one of its most characteristic features, the explanation of which has already been given. The artery rises and falls under the finger with an unusual amplitude and suddenness of expansion and recoil, the so-called water-hammer or Corrigan pulse. The sphygmomanometer shows that there is a corresponding sharp rise and fall in blood pressure, the systolic being unusually high, the diastolic unusually low.

Diagnosis.—Slight degrees of aortic insufficiency may be overlooked. The murmur is often soft and faint, not to be heard over the usual site, but audible on careful examination along the left margin of the sternum or even at the ensiform cartilage.

The hypertrophy of the heart and the rapid bounding pulse of exophthalmic goitre sometimes suggest aortic insufficiency. The capillary pulse may also be seen in this condition. The characteristic murmur is, however, absent and the second aortic sound is accentuated.

Prognosis.—Aortic insufficiency is a severe lesion, but inasmuch as the strain falls directly on the strong left ventricle it is often well borne for years. On the other hand, where it is produced by a chronic and progressive endocarditis the symptoms of circulatory disturbance

are often severe and the course short.   Aortic insufficiency in advanced years is especially likely to be complicated by attacks of angina pectoris any of which may be fatal.

IV. AORTIC STENOSIS

Stenosis of the aortic valve is a relatively rare lesion.   It most often occurs in association with general arteriosclerosis in men past middle life.

The stenosis is produced by thickening, adhesion and contraction of the valve.   The normal opening may be reduced to a narrow slit. Involvement of the coronary arteries and resulting myocardial changes are common.

MECHANICS.—The emptying of the left ventricle in systole is impeded, with resulting hypertrophy and dilatation.   In the effort to completely empty itself the ventricle takes more than normal time.   Systole is therefore prolonged and the pulse is often slow.   The pulse is also small, the artery contracted and hard.   Eventually the left ventricle dilates, the mitral valve becomes relatively incompetent and the usual course of broken compensation is followed.

Symptoms.—Cerebral symptoms due to inadequate supply of blood to the brain are common.   The patient suffers from headache, vertigo, or insomnia.   The pulse is often slow.   Otherwise the symptoms are those common to valvular lesions.

PHYSICAL SIGNS.—Inspection.—The apex is displaced downward and outward, but to a less degree than in insufficiency of this valve.   The precordium may be prominent and the impulse slow and heaving, or full.   Palpation confirms these evidences of hypertrophy of the left ventricle.   There is usually a marked thrill, systolic in time, felt over the aortic area and transmitted upward along the great vessels to the clavicle, possibly beyond.

Percussion.—The area of deep cardiac dulness is increased to the left and downward to a moderate degree.   Only in late stages will there be any increase to the right.   In the aged the increase in size of the heart is often obscured by pulmonary emphysema.

Auscultation.—A long, harsh, ''sawing'' murmur is heard over the sternal end of the second right intercostal space and to some extent round-about, but particularly upward over the great vessels to the clavicle.   The aortic second sound is obliterated or replaced by the diastolic murmur of incompetency.

The pulse of aortic stenosis is small, the artery contracted and hard. Often the rate is slow and irregular, especially in elderly persons.

Diagnosis.—The murmur of aortic stenosis may be simulated by roughening of the aorta without involvement of the valve.   In such cases the ventricle should be less hypertrophied, and the second aortic sound should be clear, or even accentuated.

Accidental or hemic murmurs may also be heard over the aortic area, but the absence of other signs of organic lesion should prevent mistakes.

V. PULMONARY INSUFFICIENCY

This lesion is one of the rarest forms of valvular disease. It may result from congenital malformation, from acquired endocarditis and possibly as the result of high tension in the pulmonary artery. It is therefore a possible result of dilatation and failure of the left heart, but as a matter of fact is extremely rare in that relation.

The SYMPTOMS depend upon failure of the right ventricle and congestion of the liver, and the extremities of the body should be especially marked.

PHYSICAL SIGNS.—A blowing diastolic murmur in the second left intercostal space close to the sternum and transmitted downward along its left margin, together with signs of dilatation and hypertrophy of the right ventricle, is characteristic.

VI. PULMONARY STENOSIS

Narrowing of the pulmonary valve, while a possible result of acquired endocarditis, is almost unknown in that relation. It is, however, the most common form of congenital lesion consistent with life beyond infancy. It is therefore the most common congenital lesion met with clinically. Persons with this lesion have been known to reach the age of 45 years.

The lesion is produced by a fusion of the semilunar cusps, so that all evidences of separation may be obliterated, and the orifice reduced to a diameter of two or three millimeters. The right ventricle will be hypertrophied and dilated and the tricuspid valve probably incompetent. The foramen ovale is always patent in congenital cases and the interventricular septum often defective.

Symptoms.—Cyanosis on the slightest exertion, or after eating, or even at rest, is the most striking symptom. The cyanosis is often of extreme grade, and yet the patients may be comparatively comfortable and able to get about easily. Deep cyanosis with such comparative comfort is seen otherwise only in advanced emphysema with dilated right heart or in those poisoned by large doses of coal-tar preparations. With the cyanosis there are dyspnea on exertion, cough, debility, a tendency to disturbances of digestion, and finally general anasarca.

PHYSICAL SIGNS.—Inspection.—The cyanosis is the obtrusive sign. It is especially evident in the lips and fingers, which are "clubbed"; that is, the terminal phalanges are unduly large. When the patient is lying down, the cyanosis may be but slight, but becomes marked in the upright position or on exertion. It varies greatly from time to time. There may be pulsation in the epigastrium or bulging of the precordium from hypertrophy of the right ventricle.

Palpation.—The signs of hypertrophy of the right ventricle may be confirmed, the apex may be somewhat displaced to the left, the impulse is that of the right ventricle.

A rough thrill, harshest over the pulmonic area, and transmitted

widely in all directions, but particularly upward and to the left in the line of the pulmonary artery, is felt with systole.

Percussion.—The area of cardiac dulness is increased especially on the right.

Auscultation.—The murmur in pronounced pulmonary stenosis is the loudest and harshest ever heard. It may easily be appreciated by the ear near to but not touching the chest, and in the ordinary case can be heard at any point on the chest, front or back. Like the thrill, it is loudest over the pulmonic valve and in the line of the pulmonary artery. The second pulmonic sound is absent, because of the fusion of the cusps of the valve and the low tension in the artery.

Diagnosis.—The history of cyanosis from birth, the murmur, thrill and enlarged right heart give an almost perfect clinical picture. The only condition which gives similar signs is the patent ductus Botalli. The accentuation or absence of the second pulmonic sound is the strongest point in differentiation, the sound being augmented in patency of the duct, absent in pulmonary stenosis. In case of doubt, the probability is altogether in favor of pulmonic stenosis, as much the more common lesion.

Prognosis.—Congenital lesions of the heart are nearly always fatal in early life. Pulmonary stenosis may, however, permit the patient to reach adult life, and instances of survival to the age of forty-five years are on record.

VII. TRICUSPID INSUFFICIENCY

Incompetency of the tricuspid valve from local endocarditis is extremely rare, while it naturally results (1) from dilatation of the right ventricle in the terminal stages of left-sided lesions of the heart, or (2) from obstruction to the pulmonary circulation due to emphysema, chronic interstitial pneumonia, advanced tuberculosis, and the like.

Symptoms.—Deep congestion of all the organs of the body and anasarca, as seen in the late stages of cardiac failure, are characteristic. These symptoms are superimposed upon those of the primary condition.

Physical Signs.—Inspection.—Bulging of the precordial area, pulsation in the epigastrium and the deep cyanosis, especially of the face and neck, are noticeable. General anasarca is usually present.

As with each systole of the ventricle blood is driven back into the auricle and thence into the unguarded veins of the neck, a distinct venous pulse may be seen, especially in the right external jugular, sometimes the left.

Palpation.—The pulsation of the right ventricle in the precordium and the epigastrium may be notable. The most important signs to the touch are, however, the venous pulse in the neck, and a systolic pulsation of the liver edge, explainable on the like regurgitation of blood into the hepatic veins.

Percussion.—The cardiac dulness is extended to a varying degree to the right of the sternum.

Auscultation.—A blowing systolic murmur resembling that of mitral insufficiency is heard over the lower portion of the sternum or in the 4th or 5th spaces on either side close to it.

Diagnosis.—The murmur of tricuspid insufficiency can be safely distinguished from that of a mitral insufficiency only by the accompanying signs. The location of the murmur is suggestive only.

VIII. TRICUSPID STENOSIS

Narrowing of the tricuspid orifice is too rare to be of practical importance. It has been found both as a congenital and an acquired lesion combined with some other defect. Of the latter class, 75 per cent. are in women and rheumatic fever is the usual cause.

The SYMPTOMS are practically those of tricuspid insufficiency. Polycythemia is regularly found. The PHYSICAL SIGNS may be inferred. The presystolic murmur is heard over the lower sternum or even to the right of it. A presystolic thrill may be felt in the same situation.

Diagnosis.—The lesion is difficult to distinguish from mitral stenosis or from tricuspid insufficiency.

COMBINED VALVULAR LESIONS OF THE HEART

The valvular lesions, described separately for the sake of simplicity and clearness, may be combined in all possible ways. (1) Lesions of the left heart greatly preponderate over those of the right. (2) Both mitral and aortic valves are often affected, and either valve may present the lesions of stenosis and insufficiency as well. (3) Aortic insufficiency or stenosis is more often combined with mitral insufficiency than with mitral stenosis. (4) Tricuspid lesions are more often associated with mitral than with aortic lesions. In the combined forms one lesion often dominates the clinical picture, the others being accessory only; on the other hand, the effects of one lesion may to some extent neutralize that of another. Thus mitral insufficiency with aortic insufficiency or stenosis partially relieves the strain upon the left ventricle, allowing part of it to fall upon the auricle.

Sequelæ and Complications of Chronic Endocarditis.—1. IN THE HEART ITSELF.—(1) Dilatation and hypertrophy have already been described. (2) Myocarditis of some degree is frequently associated. (3) Acute pericarditis, with fibrinous or serous effusion, is a common complication in severe or advanced cases, and always a dangerous one. (4) Changes in the rapidity and rhythm of the heart action are frequent. (a) Regularly the heart action is rapid, occasionally so rapid as to warrant the designation of tachycardia—even 200 or more to the minute. (b) Bradycardia, on the other hand, is very unusual. It occurs at times in aortic stenosis. (c) Arrhythmia is very common, especially in mitral lesions. (See page 195.) While in most instances irregularity of the pulse is of serous import, there are many cases in which compensation is thoroughly maintained with marked irregularity and intermittence of the pulse.

2. Embolism.—In chronic endocarditis thrombi may easily **form** upon the valves themselves or on the adjacent endocardium, or by reason of the impaired circulation in the distal veins. From any of these sites small or large clots may be swept into the blood stream and lodged in any of the distal arteries of the body, such as.—(a) Cerebral. Apoplectic attacks and hemiplegia follow. (b) Pulmonary, giving rise to hemoptysis and other signs of infarction. (c) Splenic, causing pain, tenderness and enlargement of the organ. (d) Renal, with pain and hematuria. (e) Femoral or brachial, leading to gangrene.

3. Nervous Complications.—Embolism of the cerebral arteries has already been mentioned. Cerebral hemorrhage may occur. Apart from these definite organic lesions, serious mental disturbance is not uncommon in advanced valvular lesions. Melancholia is the more common type, but mild delirium or maniacal excitement may develop.

4. Nutrition shows little change during the early stages, but late in the disease the patients regularly lose flesh and strength. The emaciation is often concealed for a time by edema, and appears more pronounced as the patient recovers and the edema lessens.

5. Fever is present during the exacerbations of the endocarditis. The fever may be either constant or intermittent, high or low.

6. Joint Symptoms.—As rheumatic fever is the cause of most cases of chronic endocarditis, stiffness and pain about the joints, either with or without redness and swelling, are frequently complained of.

Prognosis.—The outlook in chronic endocarditis depends upon many factors.

1. The Location and Nature of the Defect.—Any valvular lesion may be perfectly compensated and may remain so indefinitely. The mitral lesions are relatively more dangerous than the aortic, because the strain falls directly on the thin-walled auricle; especially is this true of mitral stenosis. Stenosis is in general more harmful than insufficiency. Multiple lesions complicate prognosis, because in some instances they add to the gravity of the condition, while in others one lesion offsets another and the combination is better borne than a single lesion.

2. The Age of the Patient.—Endocardial lesions in childhood are relatively grave. They are in great part rheumatic; the disease tends to recurrences with aggravation of the endocarditis; it is almost impossible to prevent children from overtaxing weakened hearts in play or ordinary exertion. Their liability to acute infectious diseases increases the danger.

3. Occupation.—Freedom from hard physical labor or severe exertion is necessary to safety. Endocarditis is therefore more serious in men than in women, and especially among those whose occupation involves hard work.

4. Temperament.—A calm and even mind is as important as physical quiet. Freedom from worry or excitement is quite as necessary as freedom from physical strain.

5. CONDITION OF ARTERIES AND KIDNEYS.—The presence of lesions of the arteries or kidneys always adds to the gravity of prognosis.

Treatment.—1. PREVENTION.—Inasmuch as chronic endocarditis results from acute infectious diseases, especially rheumatic fever or from the group of influences producing arteriosclerosis, such as alcohol, syphilis, lead poisoning, and gout, prevention must look to the reduction of infectious diseases and freedom from the toxic influences of the second group. The prevention of attacks of rheumatic fever, since this one disease accounts for more than 50 per cent. of all cases (see page 187), is therefore of prime importance. It is generally agreed that treatment, either by salicylates or alkalies, after the onset of an attack has little or no influence upon the frequency and severity of endocardial involvements.

2. (a) DURING COMPENSATION.—Avoidance of the influences likely to cause breaking of compensation (see page 191) is the prime consideration. A quiet, well-regulated life is called for, with strict observance of the rules of hygiene and health. The diet should be simple and adapted to the digestive capacities of the patient. The regular action of the bowels must be secured. Exercise must be adapted to the age and condition. Walking, bicycle riding on level roads, golf and such like amusements should be encouraged. Any exertion causing dyspnea or pain and palpitation should be avoided. Abundant sleep is a safe-guard. The use of alcohol or tobacco had best be forbidden and indulgence in tea or coffee carefully limited. Any intercurrent acute illness must be treated with care lest the established compensation be thereby impaired or broken. Medicines should be employed only for distinct indications, such as constipation, and cardiac stimulants are not called for.

(b) IN BROKEN COMPENSATION.—Treatment must be influenced largely by the degree of disturbance and its cause. The milder manifestations may be met by a limited reduction in mental and physical activity—without anything else. Indeed, many patients, especially the young and vigorous, even when evidences of serious disturbance have developed, promptly recover compensation when put to bed and reasonably dieted, without the use of cardiac remedies.

1. Rest, both physical and mental, is therefore the prime consideration. It must be graded according to the severity of symptoms. Usually rest in bed is most effective. In some of the severest cases, however, rest in bed is difficult or impossible, on account of orthopnea. The patient should then be made as comfortable as possible in an arm chair. Whatever contributes to comfort aids recovery. Peace of mind is as important as rest of body.

2. The diet must be suited to the condition of the patient. In general it must be limited in quantity and easily digestible. Distention of the stomach from any cause adds greatly to dyspnea and distress. In severe cases fluids alone can be given, and milk in some form or modification is most often successful. In the presence of marked edema or

anasarca the total amount of fluid given in twenty-four hours must be limited, and the use of salt restricted.

3. The bowels should, as a rule, be moved daily, either by laxatives or enemata. If edema is present, saline laxatives to produce copious watery discharges are indicated.

Medicines.—Digitalis in some form is regularly given for this condition. Theoretically some have argued that digitalis is contra-indicated, and likely to prove harmful in aortic insufficiency, because by slowing the heart rate it prolongs the period of diastole and therefore the reflux of blood into the left ventricle. Practically, however, trial is regularly made of digitalis even in this condition. The tincture in doses of v–xx minims every three or four hours, or the fresh infusion, ʒij–iv. Care must be taken to be sure of the freshness and potency of the preparations. Failure often results from lack of care in these respects. If digitalis fails or is not well tolerated by the stomach, other remedies may be tried:—Tincture of strophanthus in doses of v–x minims. Caffeine, 3 to 5 grains. Strychnine sulphate, gr. 1/30–1/20, or spartein sulphate, 1 to 2 grains. The double salts of caffeine, caffeine sodium salicylate or caffeine sodium benzoate may be used hypodermatically, in doses of from 1 to 5 grains.

The diuretic effect of digitalis may often be increased by the administration of theocin, 2 to 3 grains, thrice daily.

Within the last few years the use of a crystalline strophanthin intravenously has been recommended as a substitute for digitalis in these conditions. The remedy is given in doses of 1/120–1/160 gr. daily by injection into the median basilic or cephalic veins. Only one or two doses are ordinarily administered. The effect is continued by the administration of digitalis. If digitalis has already been given, the intravenous injection of strophanthin may produce serious disturbance from heart-block. (See p. 220.) Remarkable results have at times been obtained from its use.

Treatment of Special Symptoms.—1. Pain or Palpitation.—Morphine or opium may be used for the relief of severe pain or distress. The quiet and comfort obtained are often invaluable. An ice-cap may be kept continuously applied over the heart. Aconite may be cautiously used.

2. Dyspnea.—If due to pain it may be relieved by morphine. If caused only by congestion we must rely upon stimulation of the heart. Cupping the chest or the practice of deep breathing may be helpful. If hydrothorax be present, the fluid must be aspirated. If large amounts of fluid are present, it is best to withdraw only 500 c.c. at a time, lest the sudden relief of pressure lead to fatal edema of the lung.

If edema of the lungs occur we must rely upon cupping, the use of rapid stimulants such as nitroglycerine, camphor, caffein, and adrenalin, any or all of these to be used hypodermatically.

3. Anasarca.—Digitalis and other diuretics, such as theocin, caffeine, diuretin, are indicated. Diuretin may be given in doses of 10 grains

three or four times daily—the others as indicated above. Free purgation with limitation of total fluids to one or two pints and the restriction of salt (salt-free diet) are reasonable measures. The saline purgatives or purgative mineral waters may be given before breakfast in doses sufficient to cause free, watery movements.

The feet and legs, if much swollen, should be kept elevated. Bandaging or light massage is helpful. In extreme conditions free incisions through the skin of the leg, each several inches in length, may be made to produce free drainage. Southey's tubes for this purpose seem unnecessary. Care must be taken to maintain asepsis in these procedures.

4. DIGESTIVE DISTURBANCES.—Vomiting may be persistent. If digitalis is being given it must be stopped or given by rectum. Feeding should also be suspended and the stomach given a rest for 12 or 24 hours, only sips of water being allowed. Then feeding may be gradually resumed by giving milk or broth in small quantities, half an ounce once an hour or half hour, and gradually increasing. Delafield's mixture of equal parts of cream, milk and water, with cerium oxalate, 10 grains, and sodium bicarbonate 30 grains to every 6 ounces, is often well borne under these conditions. Egg albumin water and the like may be tried.

Constipation in the early stages must be treated by giving laxative foods and fruits if possible, using the simple laxatives, such as cascara sagrada, compound licorice powder, aloin, podophyllin, and the like. When edema is present, the saline laxatives are preferable. When the stomach is irritable, enemata must be relied upon.

5. INSOMNIA.—The simple hypnotics, sodium bromide, trional or veronal, may be tried. Codeine or morphine may be added in small doses. As compensation is restored, the insomnia disappears.

Physical Methods in the Treatment of Chronic Endocarditis.—Of recent years there has been a remarkable development of physical methods for the treatment of broken compensation. These methods are in general applicable only to the milder degrees of disturbance, marked by little or no edema, such as permit the patient to be up and about, or to the stage of convalescence, when edema has disappeared and the patient is able to be out of bed.

I. N a u h e i m B a t h s.—In the village of Nauheim, Germany, the waters of a number of springs containing much CO_2, calcium chloride and sodium chloride, have for many years been used in the treatment of these conditions with such success that the baths are now produced artificially and used the world over. To what the baths owe their effects, the warm water, the gas (CO_2) or the salts, or to their peculiar combination, is still an open question. At Nauheim waters which have lost part of their CO_2 and some of the contained mineral matter by ebullition and deposition are first used, later the waters as they rise fully charged from the springs. A gradual increase in the amounts of gas and salts is thus secured and at the same time the influence of the bath is augmented by lowering the temperature. Artificial baths representing as nearly as possible the natural waters are now used.

1. Weak Bath.—To 10 gallons of water at 95° F. add one pound sodium chloride, 1½ oz. calcium chloride; increase until 3 pounds sodium chloride and 4½ ounces calcium chloride are used. Duration of bath 5 to 15 minutes; temperature 95° to 90°.

2. Strong Bath.—To 10 gallons of water add sodium chloride 3 pounds, calcium chloride 4½ ounces, and (to develop the CO_2) sodium bicarbonate 2 ounces, and 3 ounces hydrochloric acid, increasing to 8 ounces alkali and 12 ounces acid. These chemicals are now supplied in appropriate packages by all large drug firms. Duration and temperature of baths varied as above.

II. E x e r c i s e s.—(1) Swedish Movements.—A carefully graduated series of muscular movements, at first entirely passive, later active and gently opposed by the operator. The movements begin with the simplest, finger flexion and extension, and progress through wrist and elbow to the shoulder, and then in like manner go over the lower extremity. In this manner the duration and power of the exercises are slowly increased.

(2) Zander Apparatus.—Machines, driven by steam or electric power, have been contrived to accomplish all these movements for the patient (passive movements) and are in use in some large cities, and in centers for the treatment of cardiac diseases.

(3) Schott Movements.—These are merely a series of graded exercises on the Swedish principle embodied in a system employed by Aug. and Th. Schott, of Nauheim, as an adjunct to the baths. Beginning with the simplest movements of fingers, hands and arms, without resistance, the exercises progress to complicated movements resisted by an attendant.

(4) Oertel Method (hill climbing).—The same principle of graduated exercise is employed in a different manner. The patient is directed to daily walk increasing distances up increasing grades, from 0° to 5°, 5°–10°, 10°–15° and 15°–20°. At certain resorts, such as Hot Springs, Va., paths are laid out on this principle, with distances and grades marked.

MECHANICAL AIDS IN THE STUDY OF THE CIRCULATION

Sphygmomanometer.—Within recent years various instruments designed to measure in terms of a column of water or mercury the pressure within the arteries have been brought into use. Many forms are now employed. The two in common use in hospital work are the Janeway and Stanton instruments (see Fig. 36).

Either of these has three essential parts: (1) The cuff lined by a pneumatic bag or pouch, in which the air-pressure is raised. (2) A cautery bulb (Stanton) or Pollitzer bag (Janeway) for inflating the cuff. (3) The manometer with its scale. In using these instruments the cuff is fastened closely about the arm, as in the illustration, the cuff inflated till the pressure within it obliterates the radial pulse, then by means of a valve the pressure is allowed to fall till the pulse is again

felt and the reading of the manometer is taken at this point. This is the maximum or systolic blood-pressure. Many factors tend to make the method inaccurate, especially the varying thickness of the arm, and the varying thickness and resistance of the arteries. There are also many transitory influences, such as the taking of food, exercise, and respiration, to cause variations in the measurements. Nevertheless the sphygmomanometer measures the blood-pressure much more accurately than the finger, and records of the blood-pressure are now essential in the study of circulatory disturbances.
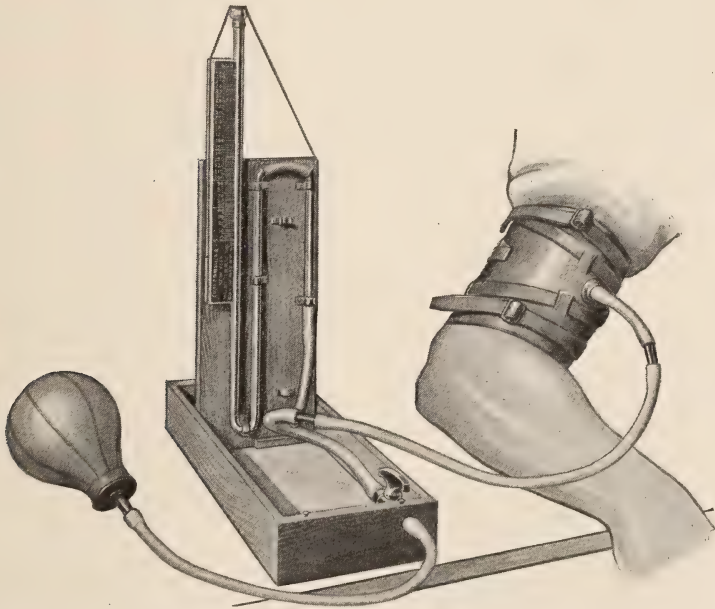
Fig. 36—Janeway sphygmomanometer, attached to arm, showing method of retention of cuff and arrangement of manometer, with Pollitzer bag inflator.

The Orthodiagram.—Taking advantage of the power of the Roentgen rays to penetrate the thorax and display the cardiac outline by its shadow, Franz M. Grödel, of Nauheim, has perfected an apparatus by which an accurate projection of the cardiac outlines is made. Much valuable information has thus been obtained of the size and shape of the heart under different conditions. The apparatus is, however, cumbrous and expensive, and involves such exposure of the operator to the rays that its use has thus far been very limited.

The Roentgen Rays.—Either by the fluoroscope or by plates the form and size of the heart may be studied. The fluoroscope is objectionable, because of the exposure of the examiner. Skiagraphic plates are valuable, but their use has been limited by the errors in projection due to varying distances of plate, heart, and Roentgen tube. The heart

shadow cannot be taken to represent accurately the size of the organ, unless the tube is at such distance as to render the rays parallel and perpendicular to the plane of the plate. Efforts to secure such results by placing the tube at a distance of 8 feet or more are now being made. Even without attention to these points the skiagraphic plates are employed to give a fairly accurate picture of the heart.

HYPERTROPHY OF THE HEART

Definition.—A thickening of the walls of one or more chambers of the heart. Hypertrophy may occur without other changes in the heart (simple hypertrophy); it is usually accompanied by enlargement in the capacity of the affected chamber (hypertrophy and dilatation or eccentric hypertrophy); very rarely it occurs with normal content or apparent reduction in the size of the chamber (concentric hypertrophy, probably only apparent and explained by systolic contraction of the affected part).

In the great majority of cases hypertrophy and dilatation, as pointed out in the last chapter, are the processes of adjustment by which compensation is brought about in chronic endocarditis, in myocarditis and adherent pericardium, and they present themselves not as independent conditions, but as attendant features of these diseases. In some cases, however, hypertrophy and dilatation occur independently of these diseases and indeed of one another, and for the sake of clearness it is necessary to study them separately.

Etiology.—The cause of hypertrophy in the heart muscle, as in any other striped muscle, is overwork. The hypertrophy appears particularly in the thick-walled ventricles, and in left or right according to the location of the strain. The auricles are less often and less strikingly affected. Not infrequently all the chambers are involved.

Hypertrophy of the left ventricle may result from (1) Excessive muscular exercises. Athletes, race-horses, and the like, regularly have hearts larger than the average, yet the degree of hypertrophy thus caused is moderate. (2) From heart lesions, such as aortic stenosis or insufficiency, or mitral insufficiency, chronic myocarditis, pericardial adhesions. (3) From overactivity or palpitation due to excessive thyroid secretion (exophthalmic goitre), the persistent use of alcohol, tea, coffee or tobacco, and the like. (4) From increased arterial tension, due to arteriosclerosis, Bright's disease, and the like.

Hypertrophy of the right ventricle may result from (1) Mitral lesions or remotely aortic lesions. (2) Pulmonary valve lesions (rarely). (3) Obstruction to the pulmonary circulation, such as occurs in emphysema or interstitial pneumonia or tuberculosis. (4) Pericardial adhesions.

Hypertrophy of the auricle on the left side is due to stenosis or incompetency of the mitral, on the right to like conditions of the tricuspid valve.

Morbid Anatomy.—For the most part the lesions are those of the underlying conditions. In the heart itself the wall of one or another

chamber is thickened, especially of the ventricles. The greatest hypertrophy is seen in the left ventricle, which may be two or three times its normal thickness. The size and weight of the whole heart is increased.

Symptoms.—Under ordinary conditions there are no symptoms, the condition being compensatory. If symptoms do appear, these are more likely to result from the underlying cause of the hypertrophy than from the hypertrophy itself. Precordial uneasiness, or discomfort, or palpitation, sometimes insomnia, may possibly be due to hypertrophy alone.

HYPERTROPHY OF THE LEFT VENTRICLE

Physical Signs.—Inspection.—Prominence of the precordium, especially in children. Apex displaced downward and outward, in sixth or seventh interspace. Impulse forcible, possibly heaving and wide. Palpation confirms the location and character of the impulse. Percussion shows the enlargement in the area of deep dulness, to the left. Auscultation.—The first sound is low-pitched and prolonged, the second aortic sound accentuated or reduplicated.

HYPERTROPHY of the RIGHT VENTRICLE gives rise to prominence of the lower sternal region in children, an epigastric pulsation, and accentuation of the second pulmonic sound.

The pulse is not materially modified unless it be by the influences producing the hypertrophy.

RADIOGRAPHIC EXAMINATION shows the increase in the size of the ventricles, either right or left, and also, indeed, of the auricles. It is especially valuable in cases of emphysema, where the distention of the lungs obscures the normal heart dulness.

Diagnosis.—From nervous palpitation, the condition may be distinguished by accurately determining the size of the heart. Unusual exposure of the heart due to retraction of the lung and caused by chronic pleurisy or interstitial pneumonia may confuse one. Here again the exact determination of the size of the heart is important. The sounds also are normal. Particular care is necessary in emphysema.

Prognosis depends entirely upon the cause and upon any signs of dilatation.

Treatment.—During compensation, treatment must be wholly directed to the cause of the condition and the avoidance of any causes of disturbance (see p. 191). When compensation fails the treatment is that of broken compensation.

DILATATION OF THE HEART

Definition.—Enlargement of one or more of the chambers of the heart. If previously normal the walls become thin; if hypertrophied, they may remain thick or appear of normal thickness. Hypertrophy and dilatation are usually associated.

Etiology.—The causes of dilatation may be grouped under two heads: (a) weakening of the walls of the heart, or (b) increase of pressure within the heart (see Rupture of Compensation, p. 191).

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Usually the two factors are combined. Dilatation tends of itself to increase. The larger any chamber of the heart becomes, the more blood it holds, the heavier its work, and therefore the greater the strain put upon it.

Morbid Anatomy.—Any or all the chambers of the heart may be enlarged. Usually there is more or less hypertrophy accompanying the dilatation. If the ventricles are dilated, the mitral and tricuspid valves become relatively incompetent, rarely the aortic or pulmonary. The endocardium is opaque and the myocardium regularly shows more or less marked degenerative changes. Arteriosclerosis and chronic Bright's disease are commonly present.

Symptoms.—As already pointed out, dilatation and hypertrophy are the protective measures by which the heart meets defects such as those of chronic endocarditis, myocarditis, arteriosclerosis, etc. In the early stages, therefore, dilatation produces no symptoms, but rather prevents those consequences which might otherwise follow valvular or other defects. In time, however, dilatation reaches the point where the circulation can no longer be properly maintained and signs of cardiac distress and progressive venous stagnation appear. The symptoms are therefore those given under rupture of compensation. In acute dilatation such as occurs during some of the acute infectious diseases the symptoms develop suddenly, it may be in a moment. Usually they are gradually evolved, covering some weeks of increasing distress.

Once dilatation has occurred the prospect of re-establishing compensation depends largely upon the cause of the dilatation and the part of the heart involved, the ventricles much more readily than the auricles recovering power.

Usually, as indicated under valvular lesions, compensation is restored and the patient lives for a varying length of time, till conditions again arise disturbing compensation. The picture of cardiac failure then develops anew. Such recurrent attacks are likely to end in one too severe to be recovered from.

PHYSICAL SIGNS.—Inspection shows a diffuse but feeble pulsation, or none at all. The apex impulse is faint or indiscernible. Evidences of hypertrophy may be seen in the prominence of the precordium. Palpation confirms these findings. Percussion gives a great increase in the area of cardiac dulness either to left or right or both, depending upon which chambers are involved. The area of cardiac dulness is still of normal form and the acute cardiohepatic angle is preserved.

Auscultation discloses the murmurs of mitral, and possibly tricuspid insufficiency. These may be accompanied by pre-existing aortic murmurs. If the heart's action is very feeble, there may be no audible murmurs and the heart sounds may be so faint as to be heard with difficulty.

The pulse is rapid, small, weak and probably irregular and intermittent. The patient gives all the other signs of cardiac failure—cyanosis, edema, ascites or hydrothorax, enlarged spleen, palpable and possibly pulsating liver.

Diagnosis.—Pericarditis with effusion presents a clinical picture distinguishable in some cases only with difficulty. A knowledge of the previous condition of the patient, or the fact that a dry pericarditis is known to have preceded the condition, is of the utmost value. The shape of the area of cardiac dulness in pericarditis with effusion is different from that of an enlarged heart, being much broader along the diaphragm, and the cardiohepatic angle can usually be demonstrated as normal in dilatation. The heart sounds and cardiac murmurs are more likely to be heard in dilatation than through a large pericardial effusion. The increased cardiac dulness in pericarditis, if studied carefully at intervals, is found to be progressive, while that in dilatation is more likely to remain fixed.

In severe infectious diseases such as typhoid fever, pneumonia, or sepsis, circulatory failure marked by cyanosis, a rapid, small, weak pulse, profound prostration, possibly pulmonary edema, and often terminating in death, is common. Such circulatory failure has long been attributed to failure of the heart assumed to be due to weakening of the heart muscle and dilatation of the heart.

Careful study in many such cases, however, shows that there is no increase in the cardiac dulness, that the heart sounds are clear and distinct, and that the apex impulse is definite, possibly forcible. Experimentally it has, moreover, been shown that this circulatory failure is due to vasomotor paralysis, especially of the splanchnic vessels, and not to "heart failure." The distinction is important with reference to treatment. Such vasomotor paralysis is to be met, not by digitalis or other heart stimulants of that class, but by cold applications to the abdomen (ice-coil or ice-bag) and the administration of caffeine, camphor or adrenalin, or clyses of normal salt solution.

Treatment is that given for broken compensation (see p. 203).

DISEASES OF THE MYOCARDIUM

MYOCARDITIS

(Myocardial Degeneration. Infarction of the Heart. Sclerosis of the Heart)

No subject in internal medicine is fraught with greater difficulty than that of the pathology and symptomatology of disease of the heart muscle. The lesions found differ markedly from those common in other organs and many times are of the type of degeneration rather than inflammation. They frequently appear in patients who have presented no evidences of cardiac disturbance; they are often lacking when confidently expected. The diagnosis of myocarditis is thus always attended with some degree of uncertainty.

Frequency.—Myocardial lesions are much more common than is supposed. In the study of the heart the more obtrusive lesions of the valves and pericardium long received more attention. Most cases of disease of either endo- or pericardium are accompanied by lesions of the muscle

and as English writers have pointed out the condition is neither endo-
carditis nor pericarditis, but a carditis, an inflammation of all the
structures in the heart wall.

But myocardial changes are present in many cases independently
of disease of the endocardium or pericardium, especially in arterio-
sclerosis with involvement of the coronary arteries. Acute degenera-
tions of the myocardium are also caused by the influence of many infec-
tions and toxemias. On the whole, therefore, myocarditis is a fre-
quent finding in post-mortem work and is doubtless present in many
cases in which hitherto it has been unthought of.

ACUTE MYOCARDITIS

Etiology.—(1) Acute infections are the most common causes, espe-
cially diphtheria, typhoid fever, influenza and rheumatic fever. (2)
Pyemia, gonorrhea and similar pyogenic infections may cause inter-
stitial myocarditis from the lodgment of septic emboli in the heart
muscle. (3) Embolism or thrombosis of the coronary artery, not in-
fective, may cause acute degeneration (necrosis) of the heart muscle.
(4) Certain severe toxemias, such as the toxemia of pregnancy or acute
yellow atrophy, may cause myocardial changes.

Morbid Anatomy.—The heart may be normal in size, or the muscle
may feel unusually relaxed and soft, or the chambers, especially the
left ventricle, may be dilated. The muscle may on section appear nor-
mal, but usually ill-defined patches in which it is paler, or more yel-
lowish, and cloudy in appearance are found. These patches are most
easily found in the walls of the ventricles. They are soft and easily
torn.

Microscopically the lesions found, although varying greatly, may
be classified as parenchymatous or interstitial. The lesions of the
muscle fibres proper are difficult to describe or to classify satisfactorily.
Special methods of treatment and study are often necessary for their
recognition. The muscle fibres may be swollen, stain poorly, be more
granular than normal, show abundant fatty degeneration, or hyaline
degeneration, or be broken in various ways. The interstitial tissue shows
lesions varying from minute foci of infiltration with lymphoid and
plasma cells to definite abscesses. Some degeneration of the neighboring
muscle always accompanies these changes in the interstitial tissue.

Symptoms.—These may develop during the course of an acute in-
fection, such as diphtheria or influenza, or they may follow it. In
severe cases syncope or sudden death may be the first symptom. In
milder cases the patients are pale and prostrated with great muscular
relaxation, or, if able to be up, they complain of weakness and pain and
palpitation on any exertion.

Physical Signs.—The heart impulse is feeble, the sounds faint and
the pulse is weak, rapid or irregular. In other cases the heart is dilated
and there are murmurs of insufficiency over one or more valves.

Prognosis.—The outlook is favorable when the condition is recog-

nized and properly treated. The possibility of sudden death, especially after diphtheria, must be borne in mind.

Treatment.—The early treatment of diphtheria by antitoxin is to some extent preventive. Rest, limited or absolute, according to the severity of the condition, is essential and must continue till all symptoms disappear.

Gastric distention must be avoided by careful and restricted feedings. Strychnine, camphor, caffein, may be used hypodermatically, if necessary. Digitalis is to be avoided.

CHRONIC MYOCARDITIS

Etiology.—Chronic myocarditis is in most cases an accompaniment of disease of the coronary arteries, chronic endocarditis or pericarditis. It may therefore have the causation of any of these conditions. It is peculiarly closely related to disease of the coronary arteries and etiologically to arteriosclerosis in general. Emphasis should be put upon the importance of syphilis, gout, alcohol, and age.

Chronic myocarditis is therefore a disease of adult life, and especially of men.

Morbid Anatomy.—The heart regularly shows dilatation and hypertrophy in some degree. Chronic endocarditis or pericarditis, or atheroma of the aorta and sclerosis of the coronaries are to be expected. The heart muscle is flabby, pale yellow or gray, more friable than normal, either in patches or diffusely.

Microscopically the interstitial tissue is thickened and increased and infiltrated with small round cells. The muscle fibres are more or less degenerated and replaced by fibrous tissue.

The changes in the coronary arteries are of such importance that they deserve special mention. The arteries show the usual changes of arteriosclerosis, thickening of the wall, tortuosity, degeneration of the intima and changes in the calibre of the vessel. Not infrequently the lumen of one of the branches is blocked by a thrombus or an embolus. If the circulation is only impaired, fibrosis occurs. If it is entirely shut off, necrosis may result.

We must include under the heading of Chronic Myocarditis the condition of fatty heart, although it is not inflammatory but degenerative in nature.

Fatty Heart.—Two forms of this condition are recognized: (1) Fatty degeneration, the muscle fibers showing degenerative changes with abnormal fatty deposition. This condition may result from a variety of conditions: (a) Failing nutrition from old age or cachectic states. (b) In protracted infectious fevers. (c) Pernicious anemia or phosphorus poisoning. (d) Chronic affections of the endocardium or pericardium or of the heart muscle itself, as in dilatation and hypertrophy.

(2) Fatty overgrowth. In obese persons an excessive deposit of fat is regularly found under the pericardium, sometimes almost concealing the heart muscle. In certain instances the fat not only overlies the

muscle, but penetrates into it, displacing the muscle strands and apparently replacing them. The muscle fibers may disappear and the ventricular wall show little but fat.

Symptoms.—Clinically the cases of chronic myocarditis may be grouped under three heads:

1. Sudden Death.—Myocarditis is the most frequent cause of sudden death due to internal disease. And in these cases thrombosis or embolism of the coronary artery is the common finding.

2. Failure of compensation in chronic endocarditis or in arteriosclerosis is often dependent upon myocarditis. These cases present themselves with symptoms of broken compensation, dyspnea, cyanosis, edema, etc., *and signs* of *dilatation of the heart* and run the course described under broken compensation.

3. Cases with weak, irregular heart action, often slow, and evidences of incompetency of the heart, such as feebleness or prostration, dyspnea on exertion, pain and palpitation and syncopal attacks or epileptiform convulsions, the so-called Stokes-Adams syndrome (see Arrhythmia, p. 224). These patients may continue to have difficulty and ultimately die in one of the attacks, or from dilatation with the usual signs of cardiac incompetency.

PROGNOSIS is always grave. The possibility of sudden death in any case must be borne in mind. Much depends upon the condition of the arteries and kidneys, still more upon the ability of the patient to lead the life of freedom from severe mental or bodily effort or anxiety called for.

Treatment.—The treatment must be that of the underlying condition (see Arteriosclerosis) or of failure of compensation if that condition develops.

NEUROSES OF THE HEART
PALPITATION

Definition.—Irregularity in the force or frequency of the heart action felt by the patient. Palpitation is usually associated with rapidity of the pulse, but may accompany a normal rate or even bradycardia.

Etiology.—1. Palpitation is one of the early symptoms of failing compensation and may belong, therefore, to any of the organic lesions thus far considered.

2. Palpitation may be a pure neurosis depending upon (a) neurasthenia, (b) gastric diseases, especially the nervous disturbances of digestion, (c) the abuse of alcohol, tobacco, coffee, or tea, (d) sexual excesses and diseases of the uterus or ovaries. Menstruation is often accompanied by palpitation or it may come on at puberty or the menopause. (e) Exophthalmic goitre or excessive doses of thyroid extract.

Symptoms.—The cardinal symptom is the consciousness of abnormal heart action. The heart action is exaggerated in force and either rapid or slow. The aorta, and the great arteries of the neck may throb vio-

lently. Manifold other symptoms due to the associated diseases (neuras-thenia, or valvular lesions, etc.) may be present.

Physical Signs.—If organic disease of the heart is present the physical signs of such lesion will be found. In other cases the heart apex is located within normal limits, but the impulse is commonly over-forcible. The area of dulness is not increased. The heart sounds are rapid or slow, exaggerated in force, often functional murmurs are pres-ent at the base, sometimes at the apex. The increased pulsation of the aorta and great vessels is often striking, the pulsation of the aorta some-times suggesting aneurism.

PROGNOSIS is good both as to life and health. The duration must depend upon the underlying causes and the possibility of their removal. Recovery is usually prompt under proper treatment.

Treatment.—Of first importance is the removal of the cause, espe-cially the prohibition of alcohol, tobacco, coffee and tea. This may in-clude the adequate treatment of gastric disturbances, neurasthenia, sex-ual disorders, organic disease of the heart or exophthalmic goitre. If the disturbance is limited to the heart alone it had best be treated on the principles of a neurasthenia. An out-door life, reasonable diet, mod-erate exercise and freedom from worry or anxiety, will, as a rule, promptly restore normal action. Bromide of soda, valerian, or asafetida is commonly given, but they are of value only as they affect the general nervous tone.

Assurance given the patient of the absence of any organic disease of the heart or danger of death is of the utmost value.

TACHYCARDIA

Any increase in rate of the heart action may be termed tachycardia. As a rule this designation is employed only for the severer grades of disturbance, in which the rate varies from 120 to 200 or more. Palpi-tation and tachycardia are commonly associated.

Etiology.—The causes are for the most part those of palpitation. Fever regularly gives rise to increase in the pulse rate. Fright may lead to persistent frequency. Lesions of the medulla or the pneumogastric nerve are found to explain some cases.

PAROXYSMAL TACHYCARDIA is a rare affection marked by attacks of tachycardia with intervals of normal heart action. The pulse may attain a rate of 200 or more per minute. The attacks of rapid action may last for minutes or days. They come without discoverable cause and cease as unaccountably.

Treatment.—The cause must be sought for and if possible removed. (See Palpitation.) In the idiopathic cases, rest must be required. An ice-cap should be applied to the precordium. Tincture of aconite in doses of from 2 to 5 minims may be given every three or four hours, or digitalis may be tried, although it usually fails of effect.

BRADYCARDIA—SLOW HEART

In certain families a slow pulse may be normal.   In some cases a rate near 40 to the minute persists throughout life.   Care should be taken to be sure that all the heart beats are perceptible at the wrist.   A person may have an infrequent radial pulse when the heart is beating rapidly through failure of part of the pulsations to reach the wrist.

Bradycardia appears to be normal in some conditions such as old age, the puerperal state, or hunger.   It is also seen in various other conditions, such as (a) the convalescence from acute fevers, especially pneumonia, typhoid fever, acute rheumatism and diphtheria.   The possible relation of myocardial degeneration to these conditions has been pointed out (see p. 212), but bradycardia often appears without other symptoms and passes off without consequence.   (b) Diseases of the digestive system, chronic gastritis, ulcer or cancer of the stomach, chronic jaundice.   (c) Diseases of the heart, especially myocarditis, of the fatty or fibrous type.   (d) In uremia and in poisoning by lead, alcohol, tea, coffee or tobacco.   (e) In anemia, chlorosis or diabetes. (f) In increased intracranial tension from hemorrhage, tumor, increased cerebrospinal fluid or inflammatory exudation.   (g) In a variety of other conditions, such as diseases of the skin or sexual organs.   The treatment must be directed to the primary condition.

ARRHYTHMIA

Disturbances of the rhythm of the heart have of recent years received an unusual amount of attention by reason of interest aroused by new methods of study and new conceptions of cardiac physiology.   The present views in this relation may be stated, as follows:—

I. Myogenic Theory.—The heart muscle possesses a number of more or less interdependent functions—(1) contractility, (2) rhythmicity, (3) conductivity, (4) sensibility, (5) tonicity.   According to these views the heart muscle possesses within itself certain powers which give rise to rhythmic contractions.   These start in the auricles near the great veins and are transmitted to the ventricles and stimulate them to contraction.   Under normal conditions the rate of auricular contractions determines the rate of systole of the ventricles.   The maintenance of these relations depends upon several factors.   (a) The normal rhythm of auricular contractions.   It has been established that the rhythmicity of the heart depends upon rhythmicity in sensibility to stimuli.   In this regard the muscle shows alternating periods of sensibility and insensibility.   The former includes the period from just before to just after contraction, the latter the remainder of each phase.   A stimulus reaching the muscle in the sensitive periods causes a contraction, while in the refractory period no result follows.   The even beat of the ventricle depends upon the maintenance of normal rhythm in the auricle.   (b) Upon the ready conduction of impulses from the auricle to the ventricle. The anatomist has shown that the only direct connection between auricles and ventricles is a minute band of muscular fibers (bundle of His, see Fig.

37), arising in the auricular septum just beneath the foramen ovale, passing downward through the auriculo-ventricular septum, lying at this point just under the mesial leaflet of the tricuspid valve, into the interventricular septum and there breaking up into a cobweb of fine branches which are distributed to various parts of both ventricles and the papillary muscles. The integrity of this bundle is essential to the normal relation between auricular and ventricular contractions. Physiologists have demonstrated that if this path of transmission be impaired, the ventricle fails to answer to some of the auricular contractions (see Fig. 38). The failure may be partial, one in every four, three or two beats (partial heart-block), or it may be complete (complete heart-block). In the latter case auricle and ventricle are found beating
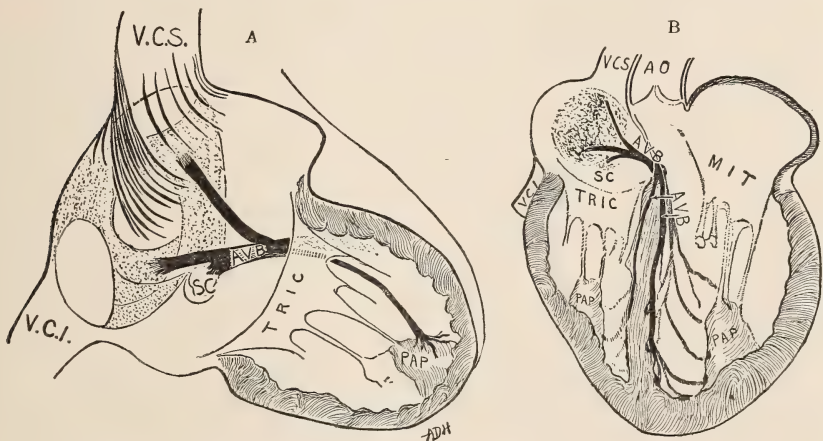
Fig. 37.—The sinus region of the heart, the veno-auricular or veno-sinal bands of striated muscle and the auriculo(atrio)-ventricular or sinu-ventricular muscle bundle. (Schematic, constructed from the findings of Keith, Schönberg, and Retzer.) A. Seen from the right side. The dotted area represents the sinus region; the striæ represent the veno-auricular muscle strands. V. C. S., superior vena cava; V.C.I., inferior vena cava; SC, coronary sinus; A V B, auriculo(atrio)-ventricular muscle bundle (His bundle); TRIC, tricuspid valve; PAP, papillary muscle. B. The same region seen from the front. M I T, mitral valve; AO, aorta. (Herschfelder's Diseases of the Heart.)

rhythmically, but with rates independent of one another, the auricular rhythm being about 60–72 to the minute, the ventricular 28 to 36, approximately one-half the auricular rate. (3) Upon normal sensibility to stimuli on the part of the ventricular muscle.

Over this muscular mechanism of the heart the nervous system presides with its center in the medulla, its vagus and sympathetic nerves. By these agencies the heart is made to respond to nervous influences either originating in the medulla or reflected upon it from other parts of the system. Thus by nervous stimulation the rate of the heart may be slowed (vagus) or accelerated (sympathetic).

II. Neurogenic Theory.—No such independence of the heart muscle is recognized, and the functions of the heart are attributed not to the powers of the heart muscle alone, but to the influence of certain nerve ganglia found in its walls. Thus the origin of the automatic stimuli is

found in the ganglia of Remak at the junction of the great veins and the auricles and these stimuli are transmitted not through the bundle of His but through nerve-fibers, on which certain other ganglia of Biedert are found.
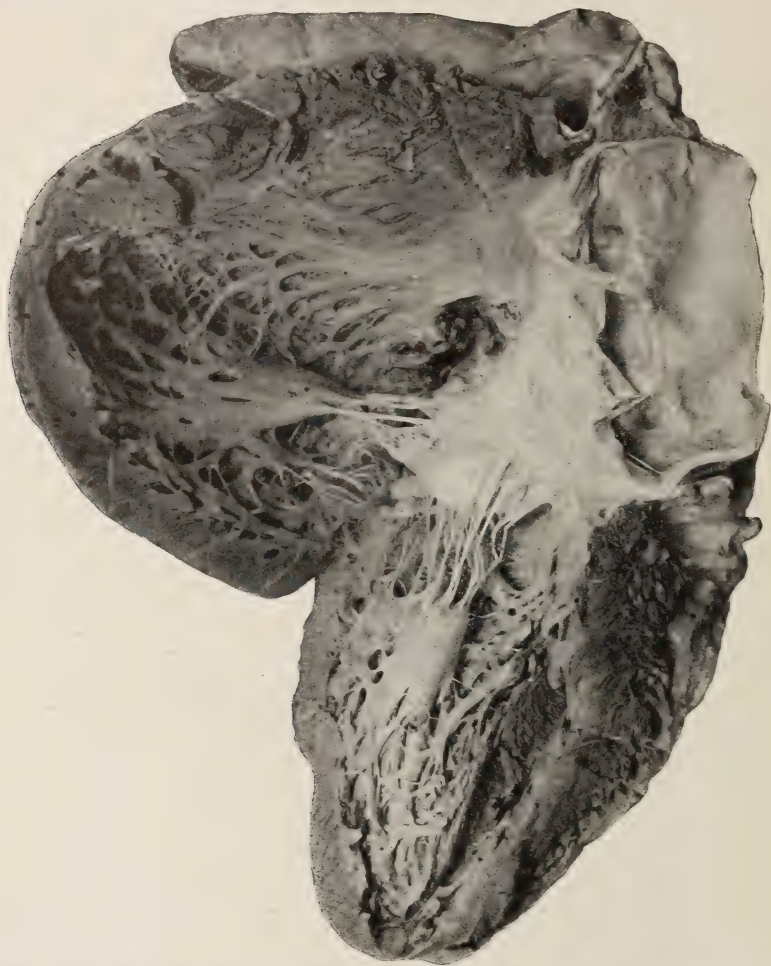
Fig. 38.—Heart showing an ulcer of the septum involving the bundle of His and producing complete heart-block. From the collection of Dr. Walter B. James.

It is not possible to accept unreservedly either theory and the final truth will doubtless prove to be a combination of both.

Methods of Examination.—Our understanding of cardiac arrhythmias has been greatly broadened by the introduction of new methods of study.

1. The Sphygmograph.—From the crude apparatus with which curious little tracings of the radial pulse used to be made, we have

advanced to more or less complex and costly apparatus permitting tracings: (a) From the wrist (radial or brachial pulse). (b) From the neck, giving a combination of the pulse waves in the carotid artery and the jugular vein. (c) From the precordium, giving the contrac-
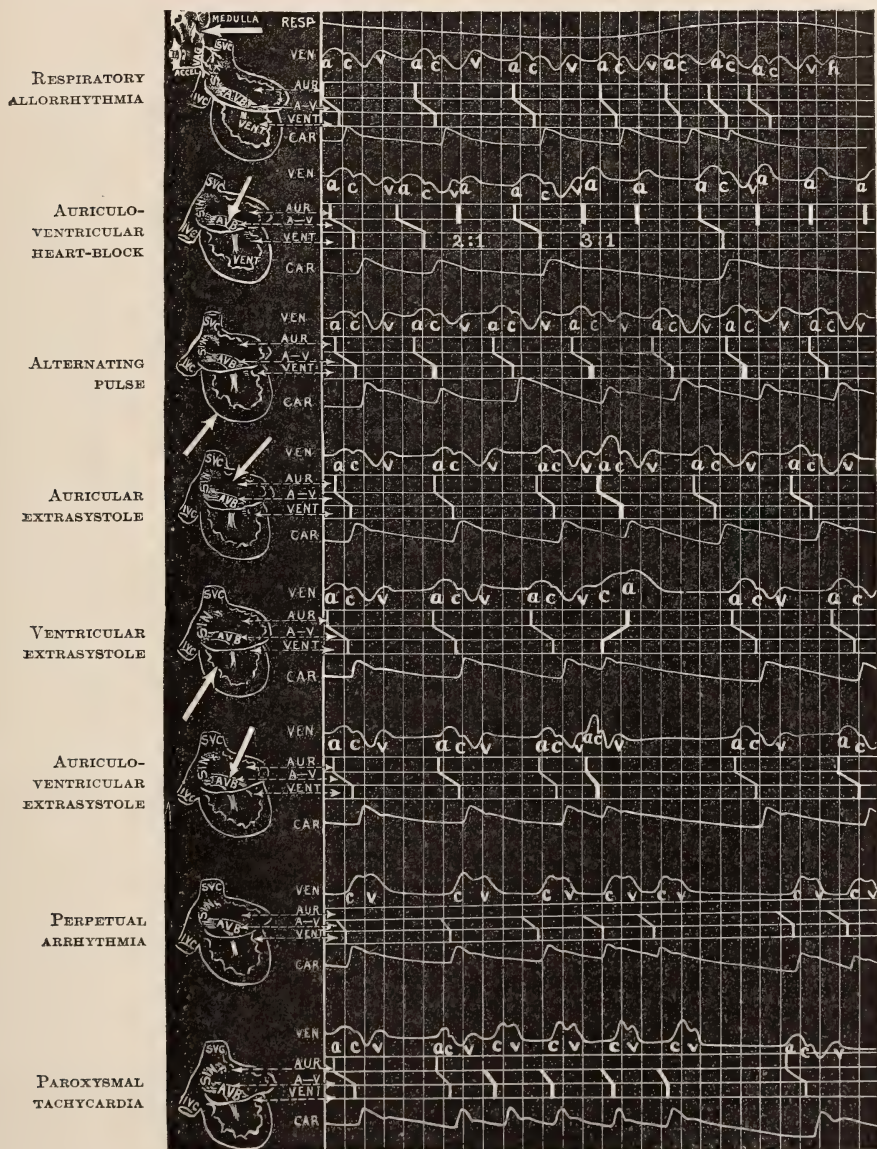
FIG. 39.—Diagram representing various types of irregular pulse. The heavy white arrows indicate the site of origin of the disturbance of rhythm. The heavy white lines indicate the course of the abnormal cardiac impulses. *RESP*, respiration; *AUR*, auricle; *A–V* or *AVB*, auriculo-ventricular bundle; *VENT*, ventricle; *CAR*, carotid pulse; *VEN*, venous pulse; *SIN*, sinus region of the heart; *SVC*, *IVC*, superior and inferior venæ cavæ, respectively. (Hirschfelder's Diseases of the Heart.)

tion of left or right ventricle according to the adjustment. (d) From the esophagus in juxtaposition to the left auricle.

Tracings from these several sources taken and compared enable one to follow quite accurately the sequence of events in the several chambers of the heart and determine their relations (see Fig. 39).

The accompanying illustrations taken from Hirschfelder's " Diseases of the Heart and Aorta " illustrate tracings from the more important types of arrhythmia. The tracings below the bars are from the carotid artery and show only the sharp rise representing ventricular systole and the slow fall. The tracings above the bars are more complicated, showing several waves, marked a, c, v. The a-wave is that produced by the auricular contraction and as can be seen by comparison with the arterial tracing just precedes the ventricular systole. The c-wave is the carotid wave, that is, a wave in the vein corresponding to the wave in the carotid artery and synchronous with it. The meaning of the v-wave is not so clear, and authorities differ in its interpretation. Mackenzie explains it as due to the storing of blood in the auricle. The crest of the wave corresponds to the opening of the tricuspid valves.

RESPIRATORY ALLORRHYTHMIA.—Quickening and slowing of the pulse occur with the phases of respiration, but each cardiac cycle is complete.

AURICULO-VENTRICULAR HEART-BLOCK.—Certain of the impulses arising in the auricle are prevented from reaching the ventricle. The corresponding auricular systoles are therefore not followed by ventricular systoles; a-waves appear in the venous pulse with no subsequent rise in the arterial tracing.

ALTERNATING PULSE (PULSUS ALTERNANS).—The systoles follow one another in regular sequence, one strong, the next weak.

AURICULAR EXTRASYSTOLE.—From time to time extrasystoles appear in the arterial tracings, each, however, preceded by an appropriate auricular systole, upon which the extrasystole depends.

VENTRICULAR EXTRASYSTOLE.—In this form the extrasystole arises in the ventricle, and is followed by a retrograde auricular systole.

AURICULO-VENTRICULAR EXTRASYSTOLE.—Here the impulse arises in tissue joining auricle and ventricle and stimulates both to contract at practically the same time instead of in the normal sequence.

PERPETUAL ARRHYTHMIA.—In this condition the auricles are said to be paralyzed, no a-waves appearing in the venous tracings and the ventricular contractions following without order, but there is evidence to show that the auricles are not inactive, but that the auricular fibers instead of contracting together in a normal manner are acting rapidly in feeble groups, fluttering or fibrillating, like the fibrillary contractions of voluntary muscle. Some of these imperfect contractions are strong enough to initiate responses in the ventricle, others are not, and the consequence is a pulse without rhythm either as to the sequence of the rises or their strength, the perpetually irregular pulse.

PAROXYSMAL TACHYCARDIA.—Two quite distinct types of pulse are commonly included under this title. In one the cardiac cycle is normal and the contraction of the ventricle follows that of the auricle in the usual manner, but the rate of action is greatly quickened, and the pulse beats number 150 or even 200 per minute. Such cases are practically excessive palpitation. In the second type the rapid action is associated with irregularity of the pulse and is usually dependent upon fibrillation of the auricules. In these cases the pulse may quickly double its rate or as quickly drop back to the usual rhythm.

2. The Electrocardiogram.—The so-called string-galvanometer of Einthoven, of Leyden, has recently played a considerable rôle in the study of the irregularities of the heart. The following explanation of the principles of the apparatus is taken from the article of Drs. W. B. James and H. B. Williams, Am. Jour. Med. Sc., Sept. and Nov., 1910.

Whenever a current of electricity passes through a magnetic field at right angles to the lines of force, it tends to be deflected to one side or the other, according to its direction. If we stretch a conductor between the poles of a magnet at right angles to the lines of force, it will tend to be deflected when traversed by a current, the direction of the deflection depending upon the direction of the current. Einthoven constructed a powerful electromagnet, through the field of which he stretched a very delicate fiber, which was either a fine platinum wire or a quartz thread which had been silvered to render it a conductor. The diameter of the finest strings measures as low as 2.5μ, or about $\frac{1}{3}$ the diameter of a red blood corpuscle, and they are invisible to the naked eye except in a strong light. The mass is so small that it is unweighable. A micrometer screw enables the operator to increase or diminish the tension of the string at will, and so vary the sensitiveness of the instrument. An arc light operating in connection with a system of condensing lenses, and an objective and projection ocular project the shadow of the string upon a screen, the varying of the distance of which enables one to secure any magnification that may be desired.

By a simple apparatus, the movements of the shadow of the string on the screen are photographed on a moving film and this preserved for study. The curves resulting are found to vary with the points of application of the electrodes. Three standard adjustments of the electrodes or leads are now employed:

   I. Right arm and left arm.
   II. Right arm and left leg.
  III. Left arm and left leg.

The normal record (see Fig. 40) is seen to present a base line which shows the position of the string when no current passes, that is, when the potential of its two ends is the same, and starting from the base line are five waves lettered P, Q, R, S, and T. The best understood and most important of these are those which rise above the base line, that is, which are positive, P, R, and T. The term positive here has
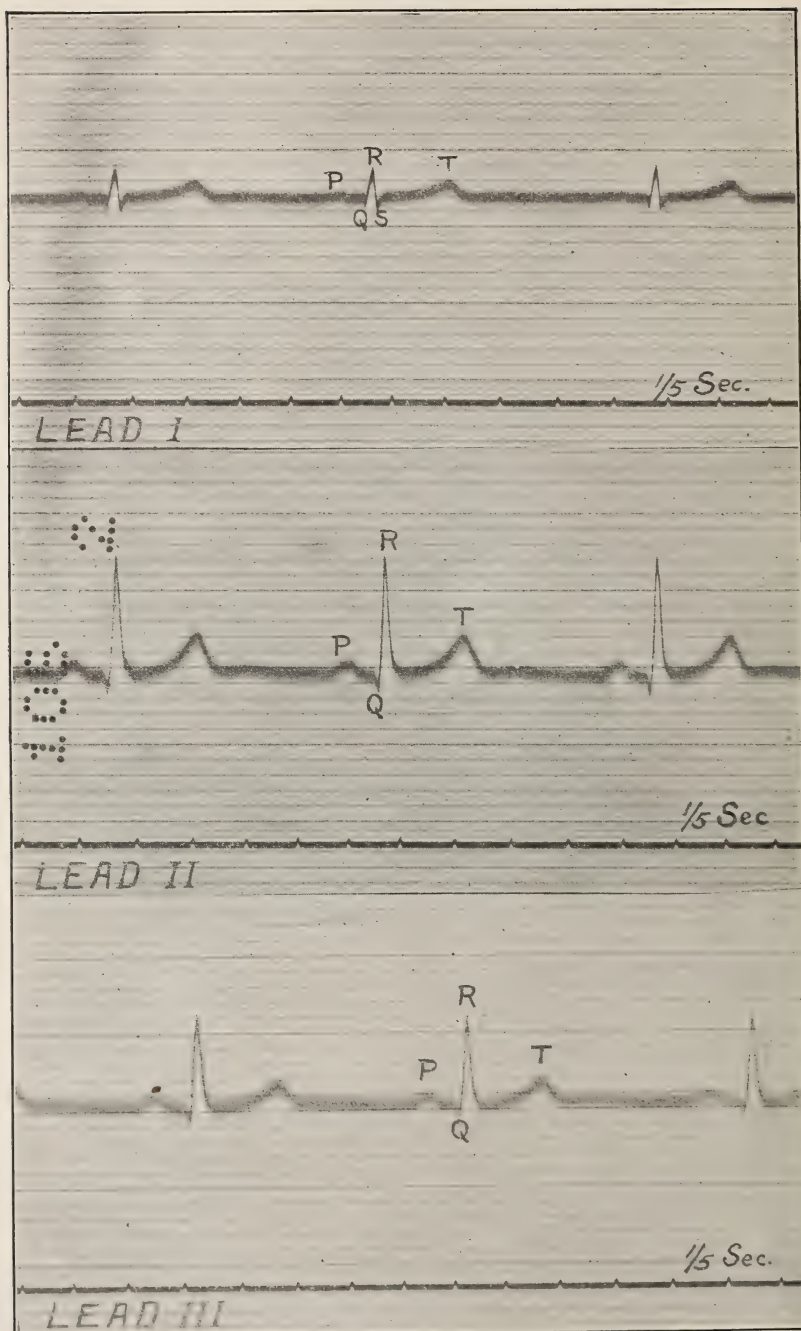
FIG. 40.—Electrocardiogram of a healthy adult male, taken while sleeping.   (Am. Jour. Med. Sci., vol. 140, page 418.)

reference only to the direction of the curve with reference to the base line. Each of these positive waves means that the base of the heart is electrically negative to the apex, or, to be more exact, that the algebraic sum of all the differences of potential that exist in the heart at that instant results in negativity of the upper end of the string toward the lower end to an extent indicated by the amplitude of the wave. P corresponds to the contraction of the auricle. The causal relation between auricular systole and P has been positively established by study of the exposed heart in animals, as well as by the observation of cases of auriculo-ventricular dissociation in human beings. Q, R, S, and T all correspond to the systole of the two ventricles. R and T are the most constant of these, and, as far as our present understanding goes, are the most important and useful in electrocardiography. R and T are always present in health, while Q and S may, either one or both, be absent.

Observers are not in entire agreement as to the significance of the waves that make up the ventricular portion of the curve, but the following is a summary of the leading views:

The space between P and Q indicates the time elapsing between excitation of the auricles and ventricles, and is, therefore, the best measure we have of the auriculo-ventricular interval.

Q directed downward, and therefore indicating negativity of the apex toward the base, has been supposed by Einthoven to be due to an initial contraction of apical fibers, and the variations in this wave in individual cases to be due to variations in the distribution of the fibers of the bundle of His. Q in health is always small and is often absent. It passes at once into R, the highest, most uniform and useful of all the waves.

R corresponds to the initial contraction of the ventricles at the base, and indicates marked negativity of the base of the heart. It is possible that it is produced by the initial contraction of the papillary muscle system. As has been shown by Hering, the papillary muscle system contracts before the apical impulse mass, a physiological fact which seems to bear out the latter view and is in agreement with the anatomical findings of Tawara, that the earliest and most extensive distribution of the fibers of the bundle of His is to the papillary muscles.

We may be reasonably sure that the wave R continues to rise while the contraction wave of the ventricle is spreading from base to apex, and the summit of R marks the instant at which this contraction has reached the apex of the heart; the time during which this wave R is rising is then a measure of the time occupied by the ventricular systole in spreading from base to apex, and may well be a matter of value in the study of diseases of the heart muscle, for, knowing the distance, it is possible to calculate the rate of travel, as has been done by Gotch in the exposed heart of the tortoise. During the interval between R and T the string remains at zero. This does not indicate a cessation of muscular activity, but only that all parts of the muscle are equally active and therefore the potential of base and apex remain the same.

It is probable that this time period also will be of value in the study of the diseased heart.

R is frequently followed immediately by a negative wave S, the significance of which is still in doubt.

T, the final wave of the ventricular phase of the electrocardiogram, is usually directed upward, and therefore indicates relative negativity of the base to the apex, that is, a preponderance of basal over apical activity at this instant, which is the very end of a ventricular systole. From the presence or absence of these several waves, their height, their time relations, much valuable information with relation to cardiac irregularities has been obtained. For the sake of comparison, Fig. 41, giving the curve obtained in a case of auricular fibrillation, is reproduced. The apparatus is too costly and too difficult of maintenance to come into general use, but in medical centers it will doubtless be found of great value.

The possibilities of this subject are indicated in the classification of cardiac arrhythmias adapted from Erlanger.

I. Arrhythmia resulting from decreased conductivity in the auriculo-ventricular junction—heart-block. A. Partial.—(1) Occasional ventricular silence. (2) Regularly recurring ventricular silence, one ventricular beat to every 7, 6, 5, 4, auricular systoles failing, or a 2 : 1, 3 : 1 or 4 : 1 rhythm prevailing. B. Complete heart-block.—Auricular and ventricular rhythm perfect, but independent. C. Paroxysmal bradycardia (Stokes-Adams disease) affecting only the ventricular rate.

II. Arrhythmia from increased irritability. A. Ventricular extrasystoles, independent systole of the ventricle, one or more following a normal systole. B. Auricular extrasystoles (including fibrillation of the auricles).

III. Arrhythmia resulting from the influence of extrinsic nerves upon the heart rate. A. Vagus effects. B. Accelerator effects.

IV. Arrhythmia resulting from disturbed diastolic filling of the heart. A. From violent respiratory movements; may give paradoxical pulse. B. From adherent pericardium or mediastinal tumor; may give paradoxical pulse. C. Associated respiratory and cardiac rhythm.

Morbid Anatomy.—While changes in the heart muscle itself, either primary or secondary to changes in the endocardium, the pericardium or other parts, naturally assume the foremost position in this relation, and most especially changes in the bundle of His (tumor, sclerosis, degenerations, etc.), it is evident that disturbances in the nervous system, the medulla, the vagus and accelerator nerves, induced either by local lesions or by changes in remote parts, such as the cerebrum (fear, fright, etc.), the digestive organs, the sexual organs (uterus and ovaries especially), may profoundly affect the rate and rhythm of the heart action.

Prognosis and treatment must be based upon our views of the causation of the arrhythmia. Extrasystoles of either auricle or ventricle are relatively harmless.
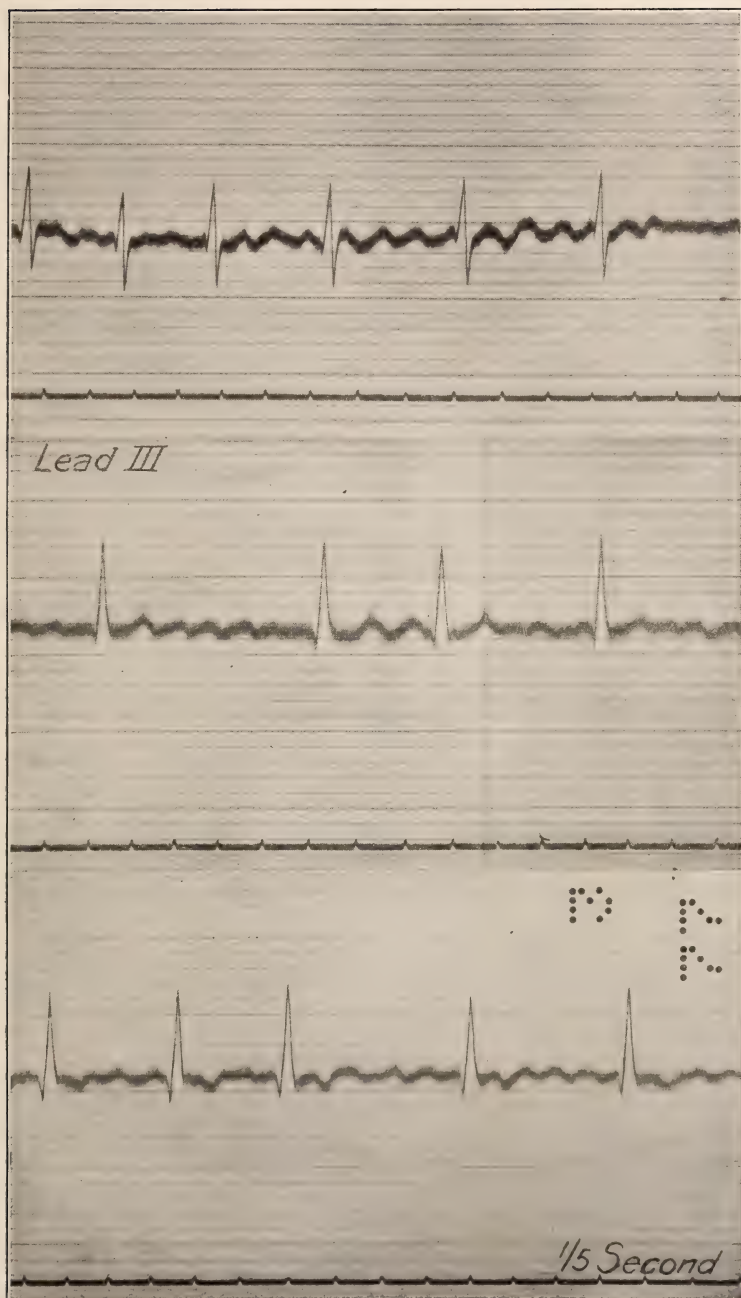
FIG. 41.—Auricular fibrillation in complete irregularity. (Am. Jour. Med. Sci., vol. 140, page 662.)

Heart-block, either partial or complete, is always serious. Remarkable instances of such conditions arising from gummatous infiltration and resulting in cure under adequate treatment are on record.

Fibrillation of the auricles is regularly an end result in failing compensation and is accordingly of grave significance. Cases are, however, known in which this condition has persisted for years. In some instances digitalis is very effective, in others it fails.

ANGINA PECTORIS

This term is employed by medical writers in two senses: (a) Etymologically as meaning and including pain of any description referred to the heart. (b) Clinically as restricted to a definite disease whose chief feature is heart pain. The latter meaning is here employed.

Etiology.—Heredity is of importance and men over 45 or 50 are more frequently affected.

The causes of arteriosclerosis, especially gout and syphilis, are commonly operative. The disease is rarely seen before thirty-five and is common only after fifty. Angina pectoris is often associated with aortic valvular lesions.

Apart from these underlying conditions of the disease the CAUSES OF THE ATTACKS OF PAIN must be considered. These may be induced by mental excitement or strain, by exposure to cold and wet, by fatigue, by attacks of dyspepsia, especially if accompanied by distention, or constipation. The use of tobacco, coffee, tea or alcohol is harmful.

Morbid Anatomy.—A variety of lesions of the heart and arteries have been found, including the following:—1. Changes in the first part of the aorta. (a) Direct injury, (b) acute aortitis, (c) atheroma, (d) aneurism. 2. Changes in the coronary vessels. (a) Acute or chronic arteritis, (b) embolism or thrombosis. 3. Lesions of the aortic valves. (a) Stenosis, (b) regurgitation. 4. Myocarditis—fatty or fibroid. 5. Degenerative changes in the cardiac ganglia. 6. In rare cases no discoverable cause.

Of these changes the most common and most important are the arteriosclerosis of the coronary vessels with secondary changes, such as thrombosis or embolism and resulting myocardial degenerations. The close anatomical relation of the aortic valves to the coronary arteries frequently leads to association of their lesions. These lesions explain the fatality attending the disease; they do not explain the attacks of pain, for they are frequently found in patients who have had no unusual pain.

The paroxysms of pain are variously explained as due (1) to cramp of part of the heart muscle, (2) overdistention of the ventricles, or (3) neuralgia of the cardiac nerves. It is not possible at present to satisfactorily decide the problem.

Symptoms.—Paroxysms of pain referred to the heart are the characteristic features. The paroxysms come suddenly, often without warn-

ing, sometimes definitely after one of the exciting causes. The pain is precordial, sharp or dull, gripping in character, often agonizing, and radiates into the neck and left arm, possibly over the whole chest. A sense of impending death accompanies it. The duration may be minutes or hours. Syncope may occur or death come in any attack.

PHYSICAL SIGNS.—The patient leans against some support, or sits down. The face becomes pale or ashen and expresses intense pain, but the patient is quiet or groans, without excitement. The pulse may show no change, or may be slow, intermittent and weak, the tension may be high or low. Cyanosis or other evidences of circulatory failure are rare.

The heart may be enlarged with an apical systolic murmur (dilatation) or may present evidences of any of the lesions of the aortic valves and coronary arteries mentioned above.

After lasting minutes, rarely hours, the pain gradually passes off and the patient is left feeling weak and exhausted. The intervals between attacks are very indefinite. They may be hours or years.

MILDER ATTACKS.—The possibility of milder attacks must be admitted, the pain being moderate and very transitory. Occurring in patients who are susceptible to severe seizures or who have definite evidences of disease of the coronary arteries or myocardium, these may be classified as angina pectoris; otherwise as pseudo-angina.

Diagnosis.—In typical form attacks of angina pectoris can hardly be mistaken. The difficulty lies in the milder cases, which may be and by some are made to include all varieties of cardiac pain. The two important questions are: (1) Have the attacks the character of true angina? (2) Are they associated with definite evidences of disease of the heart or coronary arteries? In either case they would best be regarded and treated as true angina. If both questions can be answered in the negative the diagnosis of pseudo-angina may be justified. The latter occurs in typical form in women, especially in young women, is often related to disturbance of the menstrual function, is accompanied by much excitement, often hysterical outcries, there is no evidence of arteriosclerosis or disease of the heart, the pulse is rapid and full, the face flushed. There are other evidences of hysteria or neurasthenia.

TOXIC ANGINA

Attacks of severe pain in the heart region are not uncommon from the excessive use of alcohol, tea, coffee, or tobacco. These are met with especially in young persons and are associated with palpitation. The pain is of shooting character, may be severe, may be accompanied by cold sweats and marked prostration. The attacks last several hours and may be repeated if the indulgence continues. The age of the patients, the history of overindulgence in one of the toxic substances, the absence of any signs of disease of the heart or arteries, and the results of abstinence are convincing.

PROGNOSIS must always be very guarded in true angina. Disease of the arteries and myocardium can be safely excluded only after prolonged

and careful observation. Attacks may occur without warning and any one of them be fatal. On the other hand, after one attack the patient may escape others and die of old age. Prognosis must be gravest in cases in which evidences of disease of the coronary arteries, the aortic valves, or myocardium are pronounced.

Treatment.—THE ATTACKS.—Amyl nitrite, carried in pearls containing from 3 to 5 minims, one of which is broken in the handkerchief and the fumes inhaled, is the most prompt and effective remedy. Nitroglycerine, gr. 1/100 every hour or 2 hours, or erythrol tetranitrate, gr. $\frac{1}{4}$ t.i.d., may be used if less rapid effect is desirable.

Morphine gr. $\frac{1}{4}$ with atropine gr. 1/150 may be given for the pain if there is no hypertension or the dilators do not relieve.

After the attacks rest in bed for several days must be required.

To PREVENT ATTACKS.—A quiet, even life, free from worry or anxiety, in a mild and equable climate, is indicated. (Florida or Southern California in winter, the north Atlantic coast in summer are usually chosen.) The use of alcohol, tea, coffee, or tobacco is to be forbidden or carefully limited. Underlying syphilis or gout must be treated. The diet must be simple, and suited to the patient's digestive capacity. The daily action of the bowels should be secured.

The influence of mental excitement, worry, fatigue or exposure must be avoided. Simple tonics are given as indicated. In arteriosclerotic cases iodide of potassium, 3 to 5 grains thrice daily, should be given for long periods.

CONGENITAL DEFECTS OF THE HEART

Rare even in post-mortem work in infants, congenital defects of the heart are very uncommon in clinical work, because for the most part incompatible with life beyond a few days or weeks. The following lesions are, however, occasionally met with in children or young adults:

(1) Patent foramen ovale. (2) Pulmonary stenosis (q. v.). (3) Patent ductus Botalli. Of these pulmonary stenosis is the most common and most important. The importance of patent foramen ovale, usually given as the commonest type of congenital lesion, is doubtful. It is most common, for it occurs regularly with the other lesions mentioned above, and it is met independently. So long, however, as there is no impediment to the onward passage of the blood and the two auricles contract synchronously, it is difficult to see how this defect can in any degree affect the circulation or give rise to physical signs. On the other hand, in conditions such as pulmonary stenosis it may favor the equalization of pressure in the auricles.

The SYMPTOMS of congenital lesions of the heart are given under Pulmonary Stenosis. The PHYSICAL SIGNS include cyanosis, clubbing of the fingers, enlargement of the heart and murmurs of bizarre character.

The DIFFERENTIAL DIAGNOSIS of pulmonary stenosis has already been

given. ·If the signs warrant that diagnosis, it may be advanced; otherwise we can only speculate as to the anatomical condition.

Prognosis.—Few "blue babies" survive the early months of infancy; rarely do they reach puberty. Yet they may with care do so.

Treatment.—The lines of treatment for acquired lesions must be followed. Protection from acute infectious diseases, avoidance of cardiac strain and care of the general health are most important. With signs of increasing difficulty, rest, the administration of digitalis and the other measures outlined on page 204 may be employed.

DISEASES OF THE ARTERIES

ACUTE AORTITIS

Acute inflammatory lesions of the arch of the aorta are occasionally seen as part of an acute endocarditis, particularly in lesions of the aortic valves. Very rarely indeed is the condition met with independently. In such cases it will almost surely be mistaken for aortic stenosis, the two conditions being often associated and the physical signs and symptoms being practically the same.

ARTERIOSCLEROSIS—CHRONIC ARTERITIS

Definition.—A general thickening of the walls of the arteries with degenerative changes, especially in the intima. Various terms are applied to the process. In the larger vessels it is commonly called atheroma; when the degenerative changes in the intima are unusually marked, endarteritis deformans or obliterans.

Etiology.—1. Age.—Degeneration of the arteries belongs to advancing years and in most persons over forty is present to some degree. These changes are, however, hastened and intensified by many influences.

2. Heredity.—In certain families arterial degeneration occurs early and is unusually marked.

3. Sex.—Men, more than women, are subject to the influences producing arteriosclerosis.

4. Chronic infections or intoxications, including syphilis, gout, rheumatism, diabetes. Of these syphilis is by all means most important. In this case a definite infection is present.

In the other instances chemical poisons, such as lead or uric acid, are concerned. Alcohol and tobacco are regularly enumerated under this head, but there is no agreement as to their influence.

5. Conditions causing persistent high arterial tension. (a) Continued hard muscular work. The laboring man often shows early arteriosclerosis. (b) Overeating and drinking. The constant overloading of the circulation with excessive amounts of food material, even in teetotalers, results in harm from high pressure. (c) The stress and strain of modern life seem to bring about high tension independently of other

influences.   (d) Chronic Bright's disease.   High tension is often asso-
ciated with chronic nephritis, especially the interstitial type.   Either
may be the primary affection.

Morbid Anatomy.—The lesions of arteriosclerosis are usually wide-
spread, affecting more or less evenly all the arteries of the body, and
showing especially in the aorta.   In rare instances the lesions are un-
evenly distributed and we find the arteries of particular territories
showing marked changes while others are but slightly involved.   In like
manner the individual vessel usually shows changes in all its coats, but
in most cases the changes in the intima and median coats are most im-
pressive.   Several types of arteriosclerosis may be distinguished.

1. Circumscribed or nodular form, best seen in the aorta and larger
arteries.   In the early stages small yellowish placques or nodules appear
in the intima, slightly raised, smooth.   Later the placques become larger,
more raised, then ulcerated, the intima being destroyed, and finally
infiltrated with lime, forming more or less extensive areas in the wall
of the vessel with an excavated, rough, irregular surface, of almost stony
hardness.   Such areas may quite surround the lumen of the vessel.   The
surface may show small thrombi.   The vessel wall is distorted in form,
thickened by connective-tissue formation or thinned by the necrosis and
ulceration and dilatation, and may be the seat of aneurismal sacs.

Microscopically the vessels show a round-celled infiltration or
necrosis in the media, especially about the vasa vasorum, increase in
connective tissue, fatty degeneration of the intima and of the muscular
coats.   The elastic layer also is thinned and degenerated.   The primary
changes are those of the median coat.

The result of the circumscribed arteriosclerosis is to produce in the
walls of the aorta and greater arteries localized areas of weakness and
distortion, which may become dilatations or even ruptures, which may
give rise to thrombi and infarctions or which may seriously disturb the
circulation directly or indirectly by involving the mouths of branch
vessels.

2. Generalized Arteriosclerosis.—In this process the arteries through-
out the body are more or less evenly involved.   The gross changes are
greater thickening of the walls of the vessel (so that arteries like the
radial become palpable) and greater tortuosity.   On opening the thick-
ened vessels they may appear normal, or the lumen may be distorted, or
minute dilatations may appear and the intima may here and there show
placques of degeneration such as belong to the circumscribed form.

Microscopically the most striking change is the increase in the con-
nective tissue and muscle fibers of the median coat, giving the greater
thickness to the vessel wall.   These changes are best seen in the periph-
eral vessels.   The thickened wall may be infiltrated with small round
cells and its tissues may show various degenerations.   The intima may
present areas of hyaline or fatty degeneration.   The elastic and muscular
coats are thinned and degenerate.

These changes in the smaller arteries are regularly accompanied in

some parts, especially the aorta, by the changes of the circumscribed form.

The results of generalized arteriosclerosis are thickening of the peripheral vessels with some loss of elasticity. These changes increase the resistance to the circulating blood, raise the blood-pressure and lead to hypertrophy of the left ventricle with ultimate dilatation.

The kidneys are regularly involved in the process. They are of normal size or small, the capsule is adherent, the surface somewhat rough, the cortex firm, and microscopically there is increase of connective tissue and changes more or less closely approximating those of chronic interstitial nephritis.

3. Senile Arteriosclerosis.—The walls of the larger arteries are stretched, thin, stiff and tortuous. There are degenerations of both intima and media closely resembling those seen in the diffuse process. The viscera are atrophic, the heart is not enlarged or hypertrophied, and is often smaller than normal, with thin, deep-brown muscle, the so-called brown atrophy.

Symptoms.—The relation of high blood-pressure and arteriosclerosis has been much discussed. Which is primary? Are they produced by the same cause and at the same time? At any rate they are closely associated and with thickening of the radial and other palpable arteries there is hypertrophy of the left ventricle (see p. 208) and accentuation of the second aortic sound. By this compensatory hypertrophy the heart is able to drive the blood through the narrowed and stiffened arteries and maintain for a time normal circulation. In the end the heart fails or the advance of the disease in some one territory develops symptoms relative to that part. The symptoms of arteriosclerosis may therefore be grouped as follows:

1. Cardiac.—Dilatation of the heart with all the symptoms of cardiac failure may come at any time. Dyspnea, cyanosis, edema, etc., occur as in any such failure. The heart is dilated and there are murmurs of insufficiency over the mitral and possibly the aortic valves. These cases are therefore easily mistaken for endocarditis with dilatation, but in many cases despite the signs of failure the blood-pressure is inordinately high—200 mm. or more.

Disease of the coronary arteries, fibroid myocarditis, or angina pectoris may complicate the condition.

2. Cerebral.—Headache, dizziness, insomnia, or sudden attacks of vertigo are characteristic. The cerebral disturbance may be severe. The attacks of vertigo, temporary unconsciousness, or paralysis (hemiplegia, monoplegia or aphasia) are particularly suggestive in elderly persons. These attacks may be repeated frequently and may be repeatedly recovered from, but at any time may be followed by cerebral hemorrhage and permanent disability.

3. Renal.—Some of the cases terminate as chronic interstitial nephritis and it may be impossible to determine whether the kidney or the arterial disease was primary.

4. Gangrene of the extremities (legs) may occur, especially in cases in which chronic nephritis or diabetes is present.

5. Cramps in the legs or abdomen, intermittent claudication, may appear. The cramps usually follow effort and are relieved by rest, but the reverse relation may prevail.

Course.—Once the compensation is disturbed or symptoms relative to any one part are developed, the disease is ordinarily slowly progressive and fatal in the course of a few years at most. The prognosis in any case depends largely upon the condition of the heart and kidneys and the patient's ability to lead a quiet, easy life.

Treatment.—All possible harmful influences such as alcohol, tea, coffee or tobacco should be regulated or cut off. Hard work of body or mind should be forbidden.

A moderate diet with abundance of water, and moderate daily exercise (walking, golf, driving, etc.) should be ordered. Red meats should be used sparingly and the use of fruits and vegetables enjoined. The bowels should be kept freely open and the skin active. Iodide of potassium in small doses should be given for long periods. Unless the heart is failing the high tension of the pulse is best regulated by these measures. If it must be more rapidly reduced, nitroglycerine gr. 1/100 every two hours, or erythrol tetranitrate in doses of $\frac{1}{4}$ to $\frac{1}{2}$ grain thrice daily may be tried. Often all these remedies fail.

Special symptoms relating to brain, heart or kidney must be treated as required.

Patients with arteriosclerosis do well to spend their winters in the milder climates of the south or southwest, their summers at the north. Often residence for a time at one or another of the various European spas is beneficial.

ANEURISM

Definition.—A circumscribed dilatation of an artery. A number of forms of aneurism are described. The true aneurism only is here considered. (1) A true aneurism is one whose sac is formed by one or more of the coats of an artery. (2) A false aneurism is in reality a hematoma lying in connection with a vessel but surrounded only by connective tissue, not the walls of the artery. (3) A dissecting aneurism is a dilatation of an artery by blood lying within the sheaths of the vessel itself. Such dissecting aneurisms are found chiefly in the thoracic or abdominal aorta. (4) A miliary aneurism is simply a minute dilatation of an artery, most often seen in the cerebral vessels. (5) Arterio-venous aneurisms are formed by direct communications between an artery and vein, such as the aorta and the superior vena cava. If a sac is formed between the two vessels it is called a varicose aneurism; if there is no sac and the vein is most dilated, an aneurismal varix. (6) A cirsoid aneurism is a general dilatation of an artery and its branches. They are most often seen in the arteries of the scalp. (7) Mycotic aneurisms are sometimes seen in malignant endocarditis from weakening of the wall of

an artery produced by the lodgment of infected emboli. (8) In form aneurisms are classified as sacculated, when involving only a circumscribed part of the artery, or as fusiform or cylindrical when the entire circumference of the vessel is involved.

Etiology.—Two factors are ordinarily concerned: (1) Weakening of the wall due to arteriosclerosis. Any of the causes of this condition may be operative, but syphilis is particularly effective, being present in 75 per cent. of all cases. (2) Increased blood-pressure. Any rise of pressure may cause dilatation in a weakened vessel. Heavy lifting or other severe physical exertion is often responsible for the condition. By reason of this etiology aneurisms are therefore seen most often in middle life, and in men rather than women. Congenital aneurisms are recorded, but are very rare.

Morbid Anatomy.—In the aorta, with which we have commonly to deal, an aneurism forms a tumor, in the case of the fusiform or cylindrical aneurism having the general contour of the vessel more or less enlarged, or in the sacculated form projecting from one side of the vessel and connected to its lumen by a larger or smaller orifice. Much inflammatory thickening may be present about the tumor, but the walls of the vessel itself are thinned and at autopsy often ruptured at one point. Rupture may have occurred externally through the thoracic wall, or internally into the pleura, pericardium or any of the adjacent hollow organs, the trachea, esophagus, or even stomach.

On opening an aneurism the inner surface is commonly roughened and covered by clots. Occasionally an aneurism is found in which clotting and organization of blood have occurred in successive layers till the cavity of the vessel has been more or less completely filled by connective tissue and the opening into the aorta closed (normal healing).

In other cases the original walls of the aneurism are greatly thinned, the intima more or less completely destroyed, and the roughened internal surface of the sac covered with layers of fibrin.

About aneurisms of any size there are evidences of pressure, such as erosion of sternum and ribs or even the bodies of the vertebra, compression of one or more lobes of the adjacent lung, displacement and erosion of the trachea and larger bronchi or the esophagus.

The heart is usually hypertrophied and dilated by the influences which have caused the aneurism, or it may be displaced.

The aorta and arteries show more or less arteriosclerosis and the kidneys are the seat of chronic nephritis.

Symptoms.—An aneurism is a pulsating tumor which tends to slowly but steadily enlarge. The symptoms produced by it are due to pressure and in the thorax therefore depend greatly on the position and size of the aneurism. The anatomical relations of the several parts of the aorta must be borne in mind. Aneurisms of considerable size may give no signs until rupture occurs or they may be discovered only post-mortem. Some give notable physical signs without symptoms, especially those of the ascending part of the arch, others on the

transverse arch symptoms without signs, while if placed on the descending thoracic aorta they often give neither signs nor symptoms.

Pain due to pressure is the most constant symptom. The pain is either dull or sharp, located over the base of the heart, and often radiates into the neck or arms.

Dyspnea and cough either constant or paroxysmal are usually present, owing to pressure upon the trachea or bronchi. Expectoration, usually mucous, but sometimes bloody, accompanies the cough. Otherwise the symptoms vary with the location and are best considered accordingly.

I. Aneurism of the ascending portion of the arch—the aneurism of physical signs (Broadbent).—Pain is caused by pressure
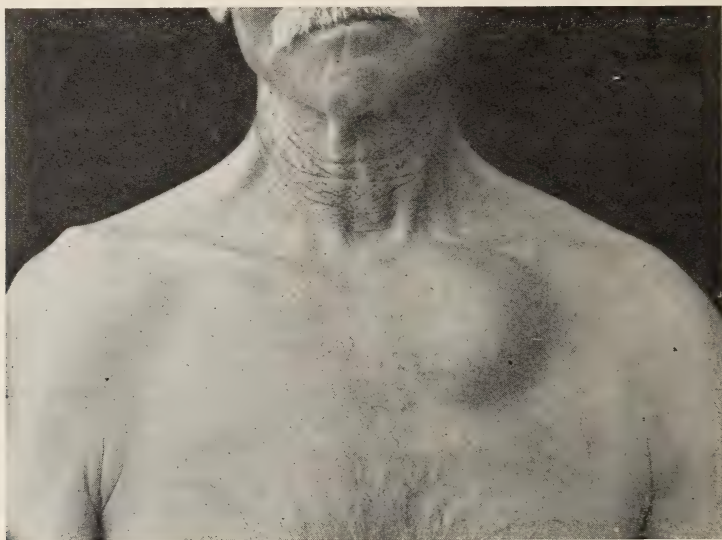
Fig. 42.—An aneurism of the arch of the aorta. From the collection of Dr. Walter B. James.

on the sternum or ribs, dyspnea by interference with the right bronchus, cardiac distress and palpitation by embarrassment of the heart action—through high blood-pressure and secondary aortic incompetency. (Fig. 42.)

Physical Signs.—(a) Bulging of the thoracic wall at or near the level of the second right costal cartilage. (b) Pulsation visible and palpable and a thrill in the first, second or third right space. (c) More or less extensive dulness in the same position. (d) Murmurs over the tumor. A systolic murmur long, loud, harsh, due to the aneurism and followed by the diastolic murmur of aortic incompetency, which can be traced downward beneath the sternum and to its left. (e) The veins of the right thorax may be enlarged by pressure on the internal mammary or the head and neck may show cyanosis from obstruction of the

superior cava. (f) The tumor may erode the thoracic wall and present itself covered only by the thin and congested integuments. It may rupture into the pleura, pericardium, or the superior cava, or through the skin.

The condition terminates in one of several ways:—

1. Rupture into the pericardium may be quickly fatal. If slow, there is precordial pain with a rapid pulse, increasing dyspnea and cyanosis. At first there are sounds resembling pericardial friction, later the area of cardiac dulness increases, the heart sounds become faint, the pulse rapid, feeble and irregular, giving the clinical picture of pericarditis with effusion. Fever may be present. 2. Rupture into the vena cava is consistent with life for several months. A sudden congestion of the head and face with cyanosis and later edema develops. Over the base of the heart is heard a *continuous* loud "boiling" murmur rising and falling with the systole and diastole of the heart and transmitted widely. There is great distention of the veins of the head, neck, arms, and upper thorax. 3. Rupture into the pleura (right) or lung will cause collapse and hemoptysis or signs of effusion in the pleura. 4. Rupture externally, death from hemorrhage. 5. Cardiac failure secondary to aortic insufficiency. This is perhaps the most common ending.

II. Aneurism of the Transverse Arch.—Aneurism of Symptoms.—1. Pain may be slight or severe. 2. Dyspnea is usually marked from pressure on the trachea and bronchi. Its paroxysmal character is notable. 3. The voice is sometimes hoarse and feeble with a brassy cough, due to pressure paralysis of the left recurrent laryngeal nerve as it winds round the aorta. 4. Difficulty in swallowing may be caused by pressure on the esophagus.

Physical Signs.—(1) The tumor presents behind or above the manubrium, or immediately to the right or left, with corresponding impulse and dulness (see Fig. 26). (2) A tracheal tug may be obtained. If the patient's head be held back so as to greatly stretch the trachea, and the lower edge of the cricoid cartilage be grasped by the examiner's fingers, with each pulsation a distinct downward tug may be felt as the expansion of the aneurism drags down the trachea.

(3) The radial pulses may be unequal, the left being smaller, and somewhat delayed, rarely it may be obliterated. The explanation may be the loss of the pulse wave in a large sac lying between the right innominate and the left subclavian arteries, or pressure upon the latter vessel or its obliteration by advanced sclerosis.

(4) Pressure on the left innominate vein may cause dilatation of the veins of the left side of the head, neck and thorax with cyanosis (see Fig. 43).

(5) Pressure on the trachea or bronchi may give rise to signs of obstruction to the entrance of air into the lung, especially on the left side. The lung may be compressed or hydrothorax follow.

(6) Pressure on the left sympathetic nerve may cause dilatation of the left pupil and flushing or sweating of that side of the face.

(7) A sharp diastolic shock due to the closure of the aortic valves under pressure may be felt if they are competent.

(8) The tumor may erode the sternum or ribs and present externally.

III. ANEURISM OF THE DESCENDING PART OF THE ARCH.—Deeply placed, close to the vertebral column, these aneurisms rarely give definite physical signs. Erosion of the bodies of the fourth and adjacent dorsal vertebræ causes pain in the back or radiating around the thorax like that of caries of the spine. The spine may be deformed as in the latter condition. The possibility of aneurism must always be remembered in adults presenting signs of caries of the mid-dorsal spine or a pressure
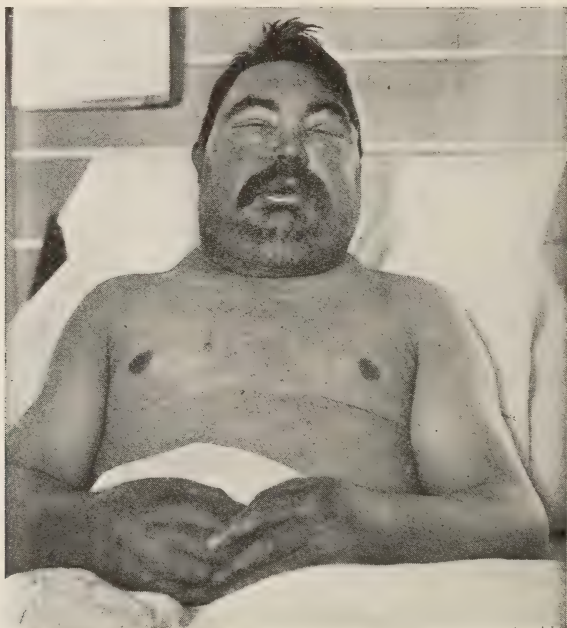
FIG. 43.—Edema of the face and arms caused by the pressure of an aneurism of the arch of the aorta.

paralysis of the lower extremities. A systolic murmur may be heard in the back and dulness and possibly pulsation observed in the back close to the left side of the fourth dorsal spine.

IV. ANEURISM OF THE THORACIC AORTA.—This differs in no essential particular from the last, except in the lower situation of the pain and the physical signs if obtained.

V. ANEURISMS OF THE ABDOMINAL AORTA.—Any part of the great trunk or of its branches may be affected, yet aneurisms of this part are rare as compared to those of the thoracic aorta. The commonest site is just below the diaphragm, where the aneurism may become large before being discovered.

The SYMPTOMS are those of tumor, pressure upon surrounding viscera or the spine.

THE PHYSICAL SIGNS.—Pulsation may be seen in the epigastrium. The tumor may be visible as well as palpable. The expansile pulsation is characteristic. A systolic murmur may be heard over the tumor, and if superficial, dulness may be obtained.

Diagnosis of Abdominal Aneurism.—In neurotic persons the aorta often displays an abnormal pulsation, and aneurism is suggested. Care must be taken to identify a tumor as distinguished from the normal outlines of the aorta.
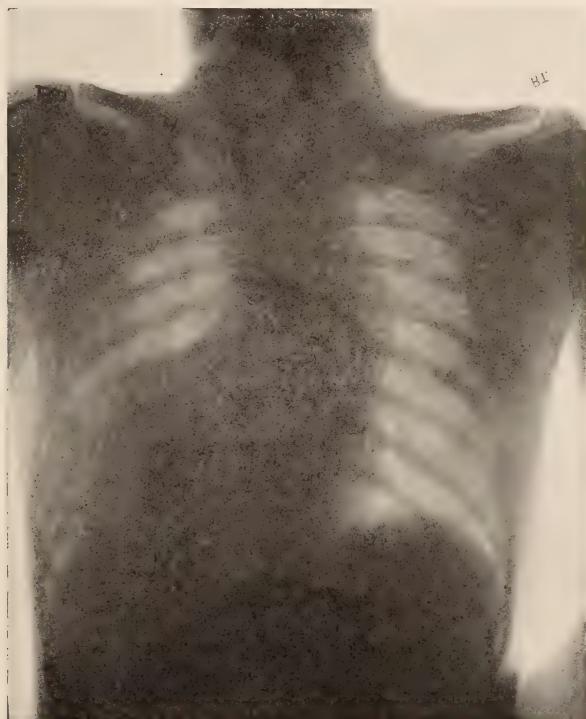
FIG. 44.—Aneurism of the arch of the aorta viewed from behind. The heavy shadow bulging to the right of the median line is the dilated aorta. Compare with the normal heart seen in Fig. 7.

Tumors overlying the aorta may pulsate, but on grasping them the expansile pulsation of an aneurism is lacking.

Ruptured abdominal aneurisms, the blood being retroperitoneal, have repeatedly been mistaken for rapidly growing, malignant tumors.

Diagnosis of Thoracic Aneurism.—Persistent pain in the chest of an adult should always suggest aneurism. Careful examination may disclose signs otherwise easily overlooked. A history of syphilis is suggestive.

Cardiac lesions, especially aortic regurgitation, give similar symp-

toms and signs. Extreme aortic stenosis may do the same. Marked enlargement of the heart is in favor of valvular lesions, but the displacement produced by aneurism may give the impression of great enlargement.

Mediastinal tumors overlying the aorta may pulsate. The thrill, murmurs and diastolic shock are absent.

Tuberculosis is frequently simulated by aneurism because compression of a bronchus may give rise to dulness and changes of voice and breathing, and vocal fremitus, while the congestion causes hemoptysis. Bacilli are absent.

Obstruction of the esophagus may suggest stricture and the possibility of aneurism must always be remembered in these cases.

Caries of the spine in an adult may be due to aneurism.

The most effective means of demonstrating or excluding aneurism of the aorta is the radiograph. No other means approaches it in accuracy. In hospitals where the X-rays are regularly employed, aneurisms are now rarely missed. (See Fig. 44.)

Prognosis.—The outlook is always grave, the duration of life after the onset of symptoms is usually not more than a few years and some cases are rapidly fatal. On the other hand, life may be prolonged for ten or fifteen years and the possibility of spontaneous healing cannot be denied.

Treatment.—Rest is the first requirement, with a light diet and restriction of fluids to a minimum. In well-nourished, vigorous men Tufnell's starvation plan may be tried. This allows but ten ounces of solid food and eight ounces of fluid per day. By these means the frequency of the heart beat and the blood-pressure are reduced while the coagulating power of the blood is increased. Calcium lactate may be given for its influence in the latter direction. Iodide of potassium, ten grains thrice daily, is commonly prescribed. The bowels must be regularly moved without straining. To be effective such treatment must be carried out for weeks or months. In most cases some amelioration is secured.

Morphine may be given for the relief of pain or dyspnea. If hydrothorax arises, frequent tapping may be required. Venesection may be employed for cyanosis.

SURGICAL METHODS.—Coagulation of blood in the sac has been promoted in a few cases by the introduction of silver wire through a fine needle passed into the aneurism. Many feet of wire have thus been reeled into the sac, and coagulation still further favored by the passage of galvanic currents through the wire. These methods have been more successful in abdominal than in thoracic aneurisms and their very limited success has not led to their general adoption.

Injections of 2 per cent. gelatin in normal salt solution, 3 ounces given every other day, with the purpose of furthering clotting have been tried. Tetanus has resulted in some instances, but can be avoided by thorough sterilization. They have but little influence.

DISEASES OF THE VEINS

ACUTE PHLEBITIS

Etiology.—Acute inflammation of the veins may occur as the result of various causes. (1) Trauma, especially in the case of the superficial veins. (2) Extension of inflammation from adjacent foci. Thus in suppurative otitis media and mastoiditis, the jugular vein may be involved; widespread inflammation of the uterine veins accompanies endometritis; about the diseased appendix or suppurative processes in the bones the veins may be extensively diseased. (3) In many of the acute infectious diseases, especially typhoid fever, septicemia, pneumonia, influenza, syphilis, and tuberculosis, phlebitis develops. In some of these cases the inflammation of the veins is explained by extension from some local focus of infection, in others the phlebitis occurs at a distance from any such foci and must be explained by the presence of organisms or toxins in the blood, and possibly by previous local injury or sclerosis of the vessel. (4) In severe anemia or cachexia phlebitis is not uncommon.

(5) Finally a certain group of cases is known in which phlebitis occurs without apparent cause (idiopathic) and in which the disturbance is attributed to gout or rheumatism. In many of these cases the existence of either gout or rheumatism is as doubtful as their relationship to the phlebitis when they are definitely established.

Morbid Anatomy.—Any or all of the coats of the vein may be involved, so that in some instances we speak of an endophlebitis, in others of a periphlebitis. Inflammation of a vein is regularly accompanied by thrombosis of varying extent. If the process be limited to a periphlebitis, thrombosis may not occur.

All grades of acute inflammation may be observed in the veins, from slight thickening due to infiltration of the walls to suppuration and complete destruction of parts of the vein. The microscopic picture varies accordingly. The thrombi are pale red or gray, adherent to the walls of the vein, and composed largely of fibrin or granular material mingled with blood cells. These thrombi may break down into abscesses, if infected, or they may become fibrous and permanently occlude the vein or be absorbed with restoration of the circulation.

Effects of Phlebitis.—If the vein remain patent, phlebitis declares itself only by the signs of local inflammation, pain, tenderness, etc.

In most cases the vein is obstructed, and the blood dammed back in the territory affected, with resulting congestion and edema. The permanency of these changes depends upon the fate of the thrombus and the efficiency of the compensatory circulation. (See Fig. 45.) In the viscera thrombosis of a vein has much the same effect as that of an artery, producing, as a rule, wedge-shaped areas of deep congestion which may suppurate, or undergo gradual transformation like an infarction (see p. 16).

In the extremities the obstruction to circulation may be temporary or permanent.

Symptoms.—GENERAL.—There are usually none, unless the phlebitis be of infective origin, when there may be a septic fever and the accompanying rapid pulse, prostration, sweating and anemia. If the phlebitis be extensive a moderate fever may be present, even in the non-infective cases.

LOCAL.—Pain, tenderness, some thickening of the vein, and possibly redness are present. The vein can usually be felt as a thickened and tender cord, especially in the extremities. Edema commonly develops in the territory drained. Thus in jugular thrombosis, edema may be
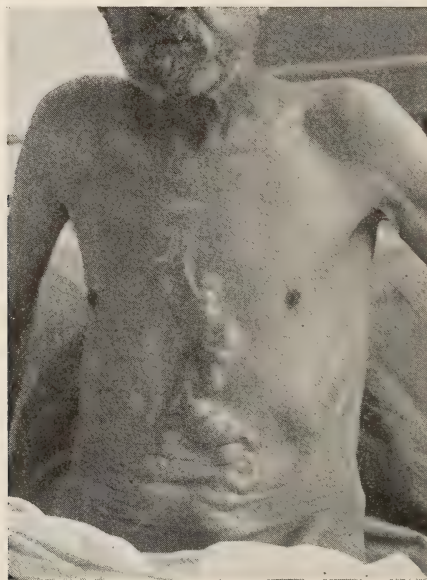
FIG. 45.—Dilatation of the superficial veins of chest and abdomen secondary to occlusion of the superior vena cava caused by pulmonary and pleural tuberculosis.

found over the mastoid and adjacent parts of the skull, and tenderness and possibly thickening of the vessel in the neck. In the lower extremities the thickened femoral or saphenous vein is easily felt, the overlying tissues may be reddened and swollen, and edema of the foot and leg below the lesion develops.

The active symptoms subside in two or three weeks, but especially in the lower extremity obstructive symptoms, swelling and edema, with some difficulty in walking, persist for months or years, depending upon the extent of the thrombosis, its permanency, and the development of compensatory circulation.

EMBOLISM.—Any thrombosis of a vein may be the source of an embolus, but fragments are particularly likely to separate from infected

and softening thrombi and thus be carried to the liver, if the primary focus lie in the territory of the portal vein, or to the lungs, if the systemic veins are concerned. Apparently minute emboli may pass through the capillaries of the lungs, reach the left side of the heart and be carried to any part of the periphery, the brain, spleen, kidneys being especially often their final lodgments. The results of such emboli depend partly upon the site in which they lodge, and whether they are infected or not. Sterile emboli produce the phenomena of infarction (see p. 16). Infected emboli may excite a suppurative inflammation or abscess where they lodge, but do not always do so. Especially is this true of the emboli derived from the heart in cases of septicemic endocarditis. The relationship of septic thrombophlebitis to pyemia is pointed out on page 375.

Special Varieties of Acute Phlebitis.—OF THE JUGULAR VEIN.—Inflammation of the lateral sinus and the jugular vein follows otitis media or mastoiditis, and is therefore regularly suppurative in character. It constitutes one of the most dangerous complications of these conditions, as from its nature it may terminate either in suppurative meningitis or pyemia.

THE SYMPTOMS are not easily differentiated from those of the primary conditions. Fever, frequently interrupted by chills, headache, vomiting and marked prostration are commonly observed. Locally there may be edema and tenderness over the mastoid, sometimes tenderness of the jugular vein in the neck, but often these local signs are wanting and the diagnosis is made only on exposure of the lateral sinus in the course of an operation on the mastoid. The presence of an old suppurative otitis media with the onset of the above symptoms, and a leukocytosis, without other discoverable cause are the important points in diagnosis. Blood cultures are usually positive. The treatment is purely surgical, the ligation of the jugular vein and removal of the infected clot found in the sinus and vein.

OF THE APPENDICULAR VEINS.—The veins of the appendix are for the most part tributaries of the portal system, but through the external or retro-colic veins communication is established with the inferior vena cava. Inflammation of these veins of varying severity occurs in appendicitis. Thrombosis follows and emboli may be carried to the liver or lungs. If the emboli be infected abscesses may result. The rapidly fatal embolism of the pulmonary artery which follows, in some instances, operations upon the appendix, is believed to have its origin in this thrombophlebitis of the veins of the appendix.

OF THE FEMORAL VEINS.—This is the most common type of thrombophlebitis. It may occur under any of the conditions given as producing phlebitis, but is especially common as a complication of the puerperium, of typhoid fever, and operations upon the abdominal organs. It is much more frequent on the left than on the right side.

The inflamed veins can usually be felt as a tender cord either imme-

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diately below Poupart's ligament or in the middle third of the thigh. Edema of the foot and leg is usually present.

Treatment.—The indications are to allay pain, limit the thrombosis, favor the circulation and provide, so far as possible, against the dangers of embolism.

The coagulability of the blood should be tested, and, if increased, efforts should be made to lower it by the free administration of water and lemon juice (citric acid). The juice of half a dozen lemons may be given daily. For the relief of pain an ice-cap should be applied to accessible veins. In the early stages morphine may be required. The circulation is to be favored by elevating the affected part, leg or arm, to aid the venous return. Absolute quiet of the affected part should be enjoined and, if necessary, enforced by the use of a splint. Ordinarily the leg or arm is made comfortable on pillows and the patient instructed to avoid all active motion.

The duration of this period of quiet varies in different cases. It must at least be maintained till all local pain and tenderness have disappeared and the danger of embolism is passed, a period of three weeks in most cases of femoral phlebitis and sometimes much longer.

Active movement of the affected part should be begun cautiously and gradually increased. If edema persists, massage may be employed, but this should likewise be used with caution and any manipulation of the affected vein or veins avoided. The wearing of an elastic stocking may eventually be necessary.

THROMBOSIS

Definition.—Thrombosis is the formation of a solid mass or plug in the living heart or vessels from the constituents of the blood (Welsh).

Etiology.—The most familiar example of thrombosis is the clot which plugs a ligated artery or vein. The injury done the intima of the vessel and the stopping of the blood stream are here the most important factors. Clinically we see many examples of thrombosis occasioned by disease of the walls of blood vessels, arteritis or phlebitis resulting in the formation of atheromatous areas in the intima (see Phlebitis, p. 239). But thrombosis undoubtedly occurs in vessels whose walls are normal, and the cause of the condition must then be sought in changes in the biochemical composition of the blood leading to its readier coagulation and to slowing of the blood stream. Under ordinary conditions the factors necessary to coagulation of the blood, thrombogen (fibrinogen) and thrombokinase (fibrin ferment), are not present as such in the blood, but are promptly developed when the blood escapes from the vessel into the tissues, for the prothrombogen of the blood then quickly becomes thrombogen and the thrombokinase is found in the vessel wall, in muscle and glandular tissues and is probably developed from the decomposition of red blood cells. What the conditions which introduce these processes within the vessels are, we do not know.

Impairment or stoppage of the circulation favors clotting within the

vessels, and thrombosis is therefore often seen in circulatory failure, especially in those forms due to disease of the heart.

Bacteria and their toxins favor thrombosis by injuring the walls of the vessels, rather than directly by their own presence, and the thrombosis thus caused is secondary to the arteritis or phlebitis.

The morbid anatomy of thrombosis is that of phlebitis except for the absence of changes in the vessel wall. Arterial thrombosis has the same results as an embolus.

The symptoms of thrombosis are those of phlebitis (see p. 240), except that the local signs are less marked. They may indeed be absent and the only evidence of the thrombosis consist of swelling and edema of the affected parts. The most familiar type of the thrombosis is seen in the brachial or axillary veins in the broken compensation of cardiac disease.

Special Types.—Thrombosis of the superior vena cava may occur, usually from pressure upon the vessels by aneurism, tumor or cicatricial processes, such as chronic pleural tuberculosis. If the process is rapid, death is immediate. If slowly developed, the obstruction is compatible with life for years. In these cases remarkable dilatation of the superficial veins of the thorax occurs (see Fig. 45).

Thrombosis of the inferior vena cava may occur, producing edema of the legs and back without ascites.

Thrombosis of the portal vein may also result from compression or from disease of the wall of the vein. This condition has been observed in a number of cases of Banti's disease, splenomegaly with recurrent ascites. The condition is most often mistaken for atrophic cirrhosis of the liver.

Thrombosis of pulmonary vessels is not to be distinguished from pulmonary infarction.

Cardiac thrombi are frequently seen, especially in septicemic endocarditis. Large thrombi are occasionally observed in the heart, especially in the auricular appendages in cases of simple chronic endocarditis. The formation of large thrombi is occasionally the immediate cause of death, but the symptoms and physical signs are too confusing to permit of diagnosis.

The treatment of thrombosis, when practicable, is that of phlebitis.

HEMORRHOIDS

(Piles)

Definition.—A localized or diffuse dilatation of the hemorrhoidal veins, especially of the anal plexus. If within the external sphincter, the hemorrhoids are spoken of as internal, as external if they involve the veins without the external sphincter. Often the two conditions are associated.

Etiology.—(1) The chief factors are mechanical, increasing the pressure within the veins. (a) The upright position naturally increases

the influence of gravity. (b) Constipation and straining at stool. (c) Stricture or other tumor of the rectum. (d) Tumors of the uterus or other abdominal organ pressing upon the rectum. (e) Pregnancy. (f) Portal obstruction of any kind, especially atrophic cirrhosis of the liver. (2) Conditions producing relaxation of the tissues concerned. (a) Old age. Hemorrhoids are rare in childhood and increase steadily in frequency with the years. (b) Debility from any cause.

Symptoms.—Piles of considerable size may give no symptoms and are often discovered in the course of routine physical examination. Disturbances of sensation, itching, burning, or pain, and hemorrhage constitute the chief symptoms of this condition. Itching or burning about the anus may produce great distress and the resultant scratching often causes eczema, erosions, or fissures of the anus. Pain is usually moderate, and is referred to the anus or adjacent parts, unless intensified by complications. These are frequent. Internal hemorrhoids are often protruded during the act of defecation and are then gripped and strangulated by the sphincter. Gentle pressure at first reduces the mass and relieves the condition, but at any time may prove ineffective. The patient suffers intense pain, radiating into the extremities, and the condition steadily grows worse, the hemorrhoidal mass becomes inflamed, swollen, possibly ulcerates. Sepsis or pyemia may result. External hemorrhoids frequently become the seat of thrombophlebitis, which causes swelling with great tenderness, so that the patient cannot sit down, and severe pain.

Constipation, one of the chief factors in the production of piles, may also result from their presence, the pain excited by defecation tending to inhibit the action.

Hemorrhage is a common occurrence from either internal or external hemorrhoids. It may vary from a slight trace of blood to considerable quantities. Bleeding most often occurs during defecation. Repeated losses of blood may occasion severe anemia.

Hemorrhoids not infrequently affect the adjacent organs, tending to produce difficulty in micturition, vaginal catarrh, and possibly disturbances of menstruation.

Prognosis.—Only in conditions of great debility or because of severe hemorrhages do hemorrhoids endanger the life of the patient. They are frequently the cause of great suffering. Often they are endured for years before the patient seeks advice regarding them. Severe anemia may be caused by repeated bleedings. Sepsis or pyemia from ulceration, while possible, is extremely rare.

Treatment.—The relief of constipation is of the first importance. The treatment of constipation must be carried out on the lines laid down on page 108. For the relief of hemorrhoids medicines that produce soft evacuations, such as salts (sodium phosphate or magnesium sulphate) or compound licorice powder, may be temporarily employed. Injections of water (Oj) or olive oil may be necessary.

Many local measures are employed. In the milder degrees sponging

the anal region with cold water after each defecation is recommended. Astringent ointments, such as the ointment of galls and opium, are often used, but are of very doubtful value. Suppositories are much more effective. The best of these are anusol (iodin-resorcin sulphate of bismuth) suppositories. For the relief of pain suppositories of opium (gr. ¼ to ½) or cocain gr. ¼ may be used. Constricted or strangulated piles may be reduced by putting the patient to bed, applying ice, and after a time gentle pressure and manipulation as in hernia. Thrombosed external piles are promptly relieved by incising them and turning out the clot. Whenever the distress caused by hemorrhoids or the bleeding is important surgical treatment should be advised. In these cases palliative measures are very unsatisfactory.

V

DISEASES OF THE BLOOD AND DUCTLESS GLANDS

ANEMIA

Definition.—Anemia signifies a reduction either of the total quantity of blood or of some of its important constituents, especially hemoglobin and the red cells. With relation to the body anemia may be local or general. Local anemia will not be discussed here. General anemia is classified as primary or essential anemia, and secondary anemia. Primary anemia includes chlorosis and pernicious anemia.

CHLOROSIS

Definition.—An anemia of young girls, without known cause, characterized by marked reduction of the hemoglobin.

Etiology.—The disease affects girls usually between 14 and 17 years of age; recurrences develop in later years. Various causes have been suggested, corsets, constipation, nervous disturbances, lack of fresh air and exercise, all of which possibly have some slight influence but cannot be regarded as determinate, since cases develop under most favorable conditions.

Symptoms.—GENERAL.—The patients are well nourished, the subcutaneous fat being preserved. The mucous membranes are pale, the skin has a pale lemon-yellow color with a tinge of green, so that the disease is often called the green sickness. Puffiness about the eyes and swelling of the ankles may suggest nephritis, while dyspnea and palpitation direct attention to the heart. Headache and neuralgia are common. The hands and feet are usually cold. Slight fever is not uncommon.

ALIMENTARY.—The appetite is capricious, often marked by a longing for pickles and like indigestible foods. Distress after meals and attacks of gastralgia are frequent. Constipation is marked.

CIRCULATORY.—Palpitation of the heart is often the symptom most complained of. Systolic murmurs over the apex of the heart and the pulmonic region commonly accompany anemia, especially the pulmonic. The heart may be slightly enlarged. The pulse is full and soft. Thrombosis may occur in the peripheral veins and pulmonary embolism may result.

UTERINE.—Amenorrhea or dysmenorrhea is commonly associated. The function returns with improvement in the blood.

THE BLOOD.—In typical cases the red cells are but little reduced (averaging 4,000,000), but the hemoglobin is low (35–50 per cent.). The index is therefore lower than in any other disease. In some cases, however, the reduction of the red cells is more marked and the blood picture closely resembles that of a secondary anemia. In severe cases

the red cells are small in size and nucleated forms may appear, but are not common. The leukocytes may be slightly increased. The total amount of the blood plasma is increased.

Diagnosis.—The anemia with a disproportionate loss of hemoglobin is characteristic. In cases resembling secondary anemia, a cause should be sought and pulmonary tuberculosis especially excluded.

Treatment.—Freedom from hard work, fresh air, and good food are required. Iron, given in the form of Blaud's pills, containing two grains of sulphate of iron, is practically a specific. One, two, and finally three pills may be given thrice daily for many weeks. Constipation must be met by laxatives such as cascara sagrada or the mineral waters. Other disturbances are treated symptomatically.

SECONDARY ANEMIA

In this form the blood condition is secondary to some primary disease or affection.

Etiology.—(1) Hemorrhage is one of the common causes, either traumatic, or resulting from gastric ulcer or cancer, cirrhosis of the liver, intestinal parasites, hemorrhoids, or like cause. The anemia is proportionate to the loss of blood. Losses of blood are quickly repaired by the normal organism, the serum and salts first, then the cells, and finally the hemoglobin. Because of this recuperative power repeated small hemorrhages are better borne than a single large one. (2) Inanition from any cause, lack of food or inability to take and digest it because of disease of the mouth, esophagus, stomach or intestinal tract, etc.

(3) Poisoning of many kinds, by the toxins of the acute infectious diseases, malaria, syphilis, typhoid fever, etc., by metallic poisons, such as lead, mercury, arsenic, copper, etc.

(4) The causes of anemia might be indefinitely extended, for nearly all the diseases included in medicine and surgery are associated, for one reason or another, with secondary anemia.

Symptoms.—The general symptoms are those of primary anemia, pallor, weakness, headache, vertigo or syncope, palpitation, dyspnea on exertion, lack of appetite, constipation, and prostration of varying degree, dependent on the severity of the anemia. Systolic murmurs over the apex or the pulmonic area of the heart may be heard.

BLOOD EXAMINATION.—The blood may be pale in color, or normal, the hemoglobin and the number of red corpuscles are reduced, but the reduction of hemoglobin is the greater, so that the index is below 1. In most cases there are but slight changes in the morphology of the red cells; in severe types the red cells become irregular in shape and size (poikilocytosis) and nucleated forms appear (normoblasts or megaloblasts), but usually in small numbers. The leukocytes are usually slightly increased.

In the restoration of the blood the serum and corpuscles are restored first, the hemoglobin but slowly returning to normal.

Diagnosis.—The examination of the blood and a knowledge of the

cause make diagnosis easy in most cases. From time to time cases of severe secondary anemia closely resembling the pernicious type are met with. If a cause can be found, such as pregnancy, infection by the hook-worm or atrophic gastritis, these cases should be classified as secondary anemias.

Treatment.—Removal of the cause of anemia if possible is necessary. Otherwise the treatment must include fresh air, rest, nutritious food and the administration of iron, arsenic and other tonics, as in primary anemia.

PERNICIOUS ANEMIA

(Idiopathic or Progressive Pernicious Anemia)

Definition.—A form of progressive anemia, marked at times by remissions, regularly fatal, characterized by an extreme reduction of the red blood cells and marked changes in their morphology.

Etiology.—The exciting cause is not known. The disease has been observed at all ages and in both sexes, but is more commonly seen in middle life, and especially in men.

Certain cases possessing the clinical characters of pernicious anemia and therefore often included in this category have been found to be due to infection with the ankylostomum duodenale or the bothriocephalus latus, or to follow pregnancy or parturition or cancer of the stomach. Although the blood picture in these cases is that of pernicious anemia, it would seem logical to transfer them to the class of secondary anemias, especially, as when the cause is recognized and removed, the condition, as a rule, is no longer either idiopathic or pernicious, but secondary and curable.

Morbid Anatomy.—The skin is extremely pale, the mucous membranes blanched. The subcutaneous fat is well preserved. The body and organs seem almost bloodless, but little pale, watery fluid exuding from the organs. Fatty degeneration of the heart, liver, kidneys and other viscera is common. Much iron pigment, the result of hemolysis, is found in the liver cells and other organs. The spleen is small and atrophic. The bone-marrow changes are important. The marrow of long bones is red throughout. Microscopic examination shows abundance of megaloblasts and evidences of stimulation of the centers from which the red cells are produced.

Symptoms.—The onset is very insidious and the disease well advanced before the patient seeks help. Debility is the most marked symptom, usually accompanied by gastro-intestinal disturbances, nausea, vomiting or diarrhea. Nervous symptoms are often marked; headache or vertigo, somnolence, irritability, blurring of vision, or tingling in the hands and feet may be complained of. The patients also suffer from the dyspnea and palpitation which belong to any anemia.

Fever of moderate degree may be present at times, but is rarely continuous.

The pallor and debility usually increase steadily, while the gastro-

intestinal symptoms are marked. Attacks of pain in the abdomen with nausea, vomiting or diarrhea are common. Minute hemorrhages into the retinæ, skin or mucous membranes are common, large hemorrhages rare.

Spinal disturbances are common. The reflexes are increased or diminished. The gait may be ataxic or weakness may prevent walking. Pains referred to the feet or legs may be present. In some cases sphincteric control is lost.

Periods of marked improvement are characteristic of pernicious anemia. These last for months or years. Death usually occurs in from one to three years, the fatal issue being preceded by general anasarca.

THE BLOOD.—The blood is very thin and watery. The red cells are reduced to an extreme degree, possibly less than 1,000,000. The hemoglobin is reduced also but not proportionately, and the index is therefore 1+. The red cells show the greatest variety in form and size. Poikilocytes, microcytes, macrocytes, nucleated forms, both large, normal and small (microblasts, normoblasts and megaloblasts), may be seen. Variations in staining are also marked (polychromatophilia). The leukocytes are reduced in number and the polynuclear percentage is low. Myelocytes are seen at times.

PHYSICAL EXAMINATION shows the pallor, possibly with minute hemorrhages in the skin, the well-preserved fat. Heart murmurs are common, usually systolic murmurs at the apex, or over the pulmonary valves, sometimes over the aortic area. Presystolic and diastolic murmurs are also heard at times. Unusual pulsation of the arteries of the neck or other parts is common. The liver may be enlarged and the subcutaneous glands palpable.

Diagnosis.—The diagnosis must rest upon the blood picture, with the absence of any cause for anemia of severe grade. The absence of intestinal parasites, particularly the ankylostomum or bothriocephalus latus, must be established, and any other cause such as malaria, syphilis, cancer and the like excluded.

Prognosis.—Recovery is possible but very rare. Periods of improvement lasting for months are not uncommon. Death usually occurs in from one to three years.

Treatment.—Rest, fresh air and nutritious food are most important. Arsenic, as Fowler's solution, is given in doses beginning with 2 drops and increasing to 10 or 15 three times daily.

Systematic colon irrigation with normal salt solution has been advocated, as a remedy for a hypothetical intestinal intoxication, and has in some cases proven beneficial. Direct transfusion of blood may prolong life for a few months when other means have failed.

LEUKEMIA

Definition.—A disease characterized by a persistent increase in the leukocytes, and associated with changes in the spleen and bone marrow or lymphatic glands.

Etiology.—The disease occurs among all races and all classes, and at all ages, but more commonly in the years from twenty to thirty. Beyond these facts nothing of real value has been determined as to the causation of the disease.

Morbid Anatomy.—The body is emaciated and extremely pale. Edema and serous effusions into the various cavities are common.

Two varieties of leukemia are recognized according to the localization of the chief lesions.

(1) Splenomyelogenous. (2) Lymphatic. It is probable that in all cases all the blood-making tissues are involved to some extent.

SPLENOMYELOGENOUS LEUKEMIA.—The *spleen* is enormous, weights of 7 to 9 pounds (4,000–5,000 grams) being recorded (see Fig. 46). The form is preserved; the surface is often covered by thickened whitish areas of perisplenitis, and adhesions are often present. The consistency and appearance of the section vary with the amount of fibrosis present.

Microscopically the organ is found to be converted into tissue closely resembling bone marrow. In the meshes of the splenic tissue are found cells of the same types as those found in the blood; myelocytes abound, then polynuclear leukocytes, neutrophiles, mononuclear cells and nucleated red cells.

Bone Marrow.—The bone marrow loses its fatty yellow appearance and becomes grayish-white, sometimes looking like pus. Microscopically myelocytes are found in abundance, numbers of non-granular mononuclears, and many nucleated red cells.

Lymphatic Nodes.—The subcutaneous lymph nodes usually show very slight enlargement, the intra-abdominal glands are larger. Microscopically some are normal, while others show more or less complete myeloid changes.

The liver, lungs, kidneys and other organs are frequently greatly enlarged by distention of the capillaries with cells of the types of those found in such numbers in the blood. The leukemic cells are often massed in rounded areas which look like tumors.

The blood presents the changes described under symptomatology.

LYMPHATIC LEUKEMIA.—The spleen is moderately enlarged and the bone marrow shows slight changes. The lymph nodes, on the other hand, are greatly enlarged and may form huge masses or tumors. The cervical group are usually most affected, but the axillary, inguinal, mediastinal and abdominal nodes are also involved.

Microscopically the nodes show an enormous increase in the lymphatic cells, and some increase in the fibrous tissue.

The bone marrow shows a similar increase in the lymphoid elements, and lymphoid infiltration is found in the other organs, the liver and kidneys especially, and sometimes in such lymphatic tissues as the tonsils, Peyer's patches, and the solitary follicles of the colon.

The blood shows changes characteristic of this form.

Symptoms.—The onset is insidious. The early complaints relate (1) to the tumor in the side, the spleen. (2) To enlargement of the

glands, especially those of the neck. (3) To anemia with its usual symptoms, pallor, dyspnea and palpitation.

Some differences develop in the course of the two types.

SPLENOMYELOGENOUS TYPE.—Usually after the onset the disease is almost stationary for months. The patients are pale and feeble; they lose flesh, from time to time they have fever of variable degree, but they complain of little except the dragging or pain due to the enlarged spleen.

Hemorrhages from mucous membranes, the nose, mouth, lungs, stomach, rectum, kidney, may occur. Retinal hemorrhage may cause blindness.

Gastro-intestinal disturbances, lack of appetite, nausea or vomiting, and constipation are common.
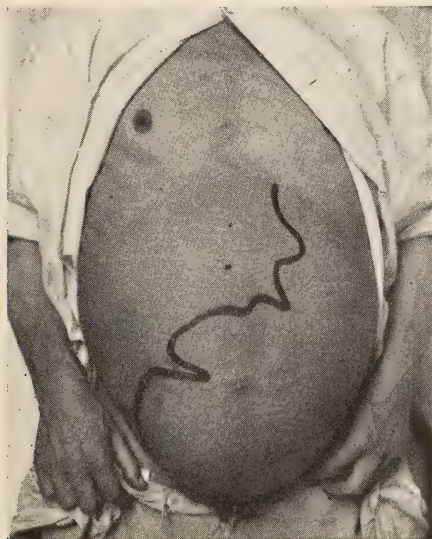
FIG. 46.—The deeply-notched anterior margin of the spleen in a splenomyelogenous leukemia.

Dyspnea and cough from leukemic infiltration of the lungs and the anemia are usually present. Hydrothorax may develop. Deafness may be caused by leukemic infiltration of the ear or hemorrhage into it. Fever of varying degree may be present throughout the course.

Finally the patients succumb to increasing anemia and feebleness, with edema and general anasarca.

The Blood.—The blood is extremely pale and watery, sometimes suggesting pus. The leukocytes are enormously increased (100,000–500,000), 1:10, 1:5 or even 1:1 of the red cells. In the differential count a considerable percentage of myelocytes, not present in numbers in normal blood, is found. They may amount to 30 to 50 per cent. of the total count. Mast-cells are also usually present, amounting to from 3 to 5 per cent.

The other forms of leukocytes normally present, polynuclears, mononuclears and eosinophiles, are present in diminished percentages which, on account of the great total, however, represent absolute increases in their numbers.

Anemia, reduction of both red cells and hemoglobin, of the secondary type, is regularly present and becomes extreme toward the close. Nucleated red cells are usually present.

PHYSICAL EXAMINATION discloses the enormous spleen, the enlarged liver, some enlargement of the lymphatic glands, so that they are palpable, possibly tenderness over the long bones, the anemia, and in the late stages hydrothorax, ascites, or edema. The heart presents hemic murmurs. The urine shows a trace of albumin and casts, but most especially a great excess of uric acid due to the breaking down of leukocytes. Gravel or stone may result.

The course is chronic, ranging from one to three years.

LYMPHATIC TYPE.—The onset and course may be the same as in the splenomyelogenous type, but cases beginning acutely and running a rapid course are not uncommon. The tumors formed by the lymphatic glands are prominent features, both in the history and physical examination. Fever may be present as in the splenomyelogenous type.

The Blood.—The leukocytosis is marked, but rarely as high as in the splenomyelogenous type, the average count being 180,000. Not infrequently the counts are low—even 20,000 to 30,000! The differential counts show the vast majority (90–95 per cent.) of these cells to be lymphocytes, sometimes the large, sometimes the small forms predominating. A few polynuclear cells are found, rarely other forms. Mast cells and myelocytes are rarely observed. The secondary anemia is present.

Diagnosis.—The disease can be overlooked by failure to examine the blood. The blood examination being made, difficulty is rare. Some of the cases of lymphatic leukemia may for a time be confused with cases of acute disease accompanied by marked lymphocytosis, such as whooping cough or miliary tuberculosis.

Prognosis.—The disease is always fatal, but the course varies, some cases surviving but a few weeks, others living two or three years. Periods of marked improvement may occur in chronic cases.

Treatment.—The usual hygienic and general measures for anemia may be employed. For the leukemia itself systematic treatment with X-rays by a skilled operator may produce marked improvement and in some cases apparent cures. The area over the spleen is usually exposed, but some operators also treat the epiphyses of long bones, the liver, or the thorax. The duration of the exposures and the intervals between them vary with individual operators, so that no rules can be given.

Arsenic is regularly given in increasing doses up to the toleration point, but is of doubtful value.

PSEUDOLEUKEMIA

(Hodgkin's Disease)

Definition.—A disease characterized by progressive enlargement of groups of lymphatic glands and by lymphoid growths in the liver, spleen, lungs or other organs, without the blood changes of leukemia.

By many the disease is regarded as a type of glandular tuberculosis, and in some cases tubercle bacilli have been found in the glands or their presence demonstrated by inoculation. On the other hand, most investigators maintain that the association with tubercle bacilli is accidental, that the bacilli are not to be found in many cases, and that the pathology of pseudoleukemia is definite and distinctive.

Pseudoleukemia is also closely related, on the other hand, to sarcoma or malignant lymphoma of the lymphatic glands, malignant new growths affecting groups of lymphatic glands and producing progressive enlargement with invasion of surrounding structures, and metastases.

Etiology.—The disease most often attacks young persons, mostly males. Seventy-five per cent. of the cases occur between the ages of ten and forty years and but few in later life. Otherwise little of value can be said as to the causation.

Morbid Anatomy.—The superficial and deep lymphatic glands are enlarged, usually in continuous chains. The enlarged glands form masses of varied size, in which, however, the individual glands are distinct; there is no fusion, no involvement of the capsule, no necrosis or suppuration. Pressure on surrounding structures is often manifest, the bronchi, veins or arteries in the chest, the ureters, veins or nerves in the lumbar region being affected.

The lymphoid tissue of the tonsils and the intestines may be hypertrophic. The spleen is enlarged, but not to the degree seen in leukemia. Lymphoid nodules are found in the spleen, liver, lungs, kidneys and other organs. The marrow of the long bones also shows lymphoid infiltration.

The blood shows a secondary anemia.

Histologically the glands in many cases show a marked fibrosis with proliferation of the lymphoid, endothelial and reticular cells and the formation of giant cells, which form the picture regarded as distinctive by Reed, Longcope and others. In other cases a simple lymphoid infiltration of the glands is found, and in still others tubercular lesions may be shown. The term pseudoleukemia must therefore still be regarded as a clinical term covering a group of conditions of diverse pathology.

Symptoms.—The onset is insidious, rarely acute. (1) Enlargement of groups of glands, progressing in the group first involved and extending to other groups, is usually the first symptom. (See Fig. 47.) The cervical are most often first affected, but the thoracic or abdominal glands may be primarily involved. As the glands enlarge the tumors produce pressure. In the neck they may cause dilatation of the veins of the head and face or congestion and edema, or dyspnea or dysphagia.

In the chest cough, dyspnea and cyanosis may result from pressure on the bronchi or great vessels, or paralysis of the vocal cords from involvement of the recurrent laryngeal. The growths are otherwise painless.

(2) Anemia, slight at first, becomes marked later, and the patients suffer from pallor, vertigo or faintness, dyspnea or palpitation as in any anemia. The blood shows a secondary anemia, without leukocytosis or the cell formula of leukemia.

(3) Cachexia.—The patients lose flesh and strength and become very feeble.

(4) Fever is often present either continuously or with periods of

Fig. 47.—The enlarged cervical and axillary lymph nodes of Hodgkin's disease. From the collection of Dr. Walter B. James.

normal temperature. It is usually slight and irregular, but may amount to a rise of several degrees daily.

PHYSICAL EXAMINATION shows: (1) The masses of enlarged glands in the neck, axillæ, inguinal regions, abdomen or thorax. Radiographs of the chest are valuable aids in diagnosis (see Fig. 39).

(2) The moderately enlarged spleen.

(3) The secondary anemia.

Course and Prognosis.—The disease is chronic and progressive, with periods of improvement in some cases. The usual duration of life is two to three years. Recovery under treatment is occasionally reported.

Diagnosis.—Leukemia is excluded by the blood examination. Tuber-

culosis must be elimininated.   (1) **By** the examination of an excised gland, histologically, and for bacilli, and finally by inoculation.   (2) By tuberculin tests, by von Pirquet's method, or by subcutaneous inoculation, if the patient is afrebrile.   (3) By the absence of tuberculosis elsewhere, especially in the lungs.

Lymphosarcoma may be confused with pseudoleukemia.   The masses are larger in this condition, surrounding structures are invaded, metastases occur in other organs, and finally the histological examination is quite different.   Lymphosarcoma is also much rarer than Hodgkin's disease.

Treatment.—Surgical removal of the glands may relieve pressure, and prolong life.   Repeated exposures to the X-rays may cause shrinking and even disappearance of local tumors.   With cessation of the treatment, growths reappear.

The administration of Fowler's solution to the point of tolerance may also be helpful.

PURPURA

(Morbus Maculosus)

Definition.—A condition characterized by the appearance of hemorrhages into the skin or mucous membranes, these hemorrhages appearing as minute spots (petechiæ) or larger hemorrhages (vibices or ecchymoses).

Hemorrhagic eruptions belonging in this category occur symptomatically under various conditions.

(1) In acute infectious diseases, especially small-pox, cerebrospinal fever, measles, septic infections, etc.

(2) In poisoning with iodine, mercury and various other drugs.

(3) In arteriosclerosis.

(4) In cachectic states, such as occur in children suffering from gastro-intestinal diseases, in Bright's disease, cancer, tuberculosis, severe anemia of any kind, etc.

(5) In conditions putting unusual mechanical strain on the capillaries, such as epilepsy or whooping cough.

All these conditions being excluded we find purpuric eruptions occurring without apparent cause and as the prominent symptom in certain cases which are grouped together as purpura.

Etiology.—Idiopathic purpura occurs at all ages, more frequently in males.   Certain families exhibit a tendency to the affection.

The disease is regarded by some as an acute infection.   In other cases the condition of the blood-vessels is of primary importance.

Symptoms.—The cases of idiopathic purpura may be grouped under three heads, representing different degrees of the affection.

(1) Purpura simplex, the mildest form, seen chiefly in children, and without the joint manifestations common in other cases.   Petechial spots or ecchymoses appear in the skin, especially on the lower extremities, the

patients are anemic and somewhat feeble, but otherwise feel well. A slight fever may accompany the eruption (see Fig. 48).

(2) Purpura Rheumatica.—Peliosis rheumatica or Schönlein's disease. In this condition the purpuric eruption is associated with swelling and pain in the joints, symptoms usually wrongly regarded as indicating "rheumatism." Fever of moderate grade (102°–103°) is regularly present. The patients are anemic and somewhat prostrated.

(3) Purpura Hemorrhagica.—In this form the purpuric eruption is accompanied by hemorrhages from the mucous membranes. Epistaxis, bleeding from the gums or mouth, hemoptysis, or hematemesis may occur, or blood may appear in the stools or urine. Fever is regularly present, the prostration is marked, the anemia severe, and occasionally the disease is fatal.

Any form of purpura may be accompanied by digestive disturbances, loss of appetite, nausea, vomiting or diarrhea, attacks of abdominal

FIG. 48.—An extensive purpura of the legs, some fresh dark spots, others fading.

pain. These symptoms are especially marked in the form known as Henoch's purpura.

A tendency to recurrence is noted in all forms of the disease.

Course.—This varies greatly. In the simple forms the eruption disappears in a few days. In the severer types the condition persists for weeks or months with recurrence of the eruption and other disturbances. In the severe or fulminating types of purpura death may occur.

Complications.—Acute nephritis and cerebral hemorrhage are not uncommon.

THE BLOOD.—A secondary anemia is usually present. The blood platelets, which usually amount to 500,000 to 600,000 per cubic millimeter, are markedly reduced, sometimes as low as 22,000. It is very remarkable that the coagulation time of the blood is usually normal or less than normal.

Estimation of the Coagulation Time.—Various methods have been employed. (1) The simplest consists in putting a drop of blood 5 mm. in diameter on a glass slide which is kept inverted over a glass of water at

40° C. and covered with a damp cloth. Coagulation is determined by turning the slide on edge from time to time and noting when the drop no longer sags. The normal by this method is 5 to 7 minutes. (2) Brodie-Russell Method.—A drop of blood on a slide is subjected to a stream of air blown upon it from a bulb, and the cessation of motion in the drop noted under the microscope.

Diagnosis.—The cases of symptomatic purpura must be excluded, especially the acute infectious diseases complicated by purpuric eruptions. The history of the patient and the results of physical examination or a few days' observation should settle the question. Cerebrospinal fever or malignant endocarditis or a septic infection should be especially looked for.

The possibility of mistaking the bites of mosquitoes or fleas for purpura must be borne in mind. The presence of the insects, the whitish areola which usually surrounds bites, and the central puncture should distinguish them.

Prognosis depends entirely upon the severity of the manifestations in the individual case. It is not often the loss of blood but the severe constitutional symptoms, or complicating nephritis or cerebral hemorrhage which causes the fatal result.

Treatment.—Rest in bed, fresh air and nutritious food are essentials. Fowler's solution is administered in increasing doses. Calcium lactate should be given, 15 grains thrice daily to an adult.

In bleeding from mucous membranes, when accessible, the solution of adrenalin chloride 1/1000 may be applied locally, or given by mouth if the bleeding is from the stomach.

In severe cases injections of normal serum, either human or animal, have been effective when other means had failed. The serum is given subcutaneously in quantities varying from 15 to 30 c.c.

Transfusion of blood from a sound person may be tried, as a last resort.

HEMOPHILIA

Definition.—An hereditary constitutional affection characterized by a tendency to severe and often uncontrollable bleedings. The hemorrhages result from trivial cuts or bruises, or occur spontaneously.

Etiology.—The affection usually manifests itself early in life, but may not appear till the twentieth year or even later. The tendency to bleed affects males particularly, but is transmitted for the most part through the female members of the family, many of whom are not themselves bleeders. The tendency is transmitted in certain families and but rarely appears in others. In some of the affected families the tendency has been found to persist for seven generations.

Morbid Anatomy.—No constant lesions other than the anemia produced by bleeding can be found in fatal cases. In some patients the arteries have been small and thin and the tendency to bleed has been attributed to this condition, but it is by no means constant. The coagula-

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tion time of the blood may be retarded to 45 minutes or even an hour, but has also been found normal in some cases.

The theory at present accepted is that hemophilia is due to chemical changes in the blood and the walls of the blood vessels, owing to which there is a deficient production of thrombokinase, one of the essential elements in the process of coagulation.

Symptoms.—The tendency to bleeding rarely appears at birth, but in most cases manifests itself before the end of the second year. One case is on record in which it first appeared in the sixtieth year. The bleeding may begin spontaneously, but usually follows some slight cut or bruise. Operations of the simplest kind, such as circumcision, may start it. Spontaneous bleeding may occur from the nose, mouth, stomach, intestines, urethra, or any other mucous membrane. Profuse uterine bleeding may occur in women with menstruation or after parturition, but this manifestation is rare. Once bleeding is begun, it may stop spontaneously, or under treatment, or end only with the death of the patient. Profound anemia naturally occurs in those who survive attacks, and there is great danger of recurrence. Petechiæ, ecchymoses, or large subcutaneous hematomata may be formed. Arthritic symptoms develop in many cases, the larger joints becoming swollen and painful, and being sometimes left stiff and deformed.

THE BLOOD.—A secondary anemia is regularly present. There is a leukopenia with an increased percentage of lymphocytes. The coagulation time is increased, as much as an hour being sometimes required for clotting.

Diagnosis.—A persistent tendency to bleeding, coupled with a family history of hemophilia, makes the diagnosis easy. It may be difficult or impossible to distinguish from chronic purpura in patients without a hemophilic family history.

Prognosis.—Children showing true hemophilia rarely reach puberty. In older patients the prognosis is less grave. The first bleeding is rarely fatal.

Treatment.—The members of hemophiliac families should be advised against marriage, especially the women. Children of such families should be protected from accident or injury so far as possible and surgical operations of any kind avoided.

For the control of hemorrhage, rest, cold, pressure and the ordinary styptics may be tried. Calcium lactate, in doses of 1 to 2 grams (15–30 grains) three or four times daily, is given. On mucous surfaces applications of adrenalin chloride 1/1000 are often effective. For bleeding from the stomach it may be given by mouth. Applications of gauze soaked in a 2 to 5 per cent. sterile solution of gelatin have also proven serviceable.

In intervals between bleedings, calcium lactate should be given as above for one or two days in each week.

Injections of normal serum or transfusion may be employed, as for purpura (see p. 257).

MORBUS MACULOSUS NEONATORUM

Bleeding from the navel or the mucous membranes is common in the new-born. The hemorrhage may be associated with the jaundice of the new-born. It begins spontaneously during the first days of life, or rarely in the second or third week. In most cases the bleeding from the mouth, the bowel, or urinary tract goes on steadily till the child is dead. Spontaneous arrest may, however, occur. The measures given under purpura are tried, but usually without effect. In desperate cases transfusion by the direct method of Carrel has been successful. Recently J. E. Welch has employed with success the serum of normal individuals, giving it in doses of 10 c.c. every two hours. The reason for the efficacy of the serum is not clear; probably it supplies some element necessary to the coagulation of the blood.

SCURVY

Definition.—A disorder of nutrition characterized by anemia, debility, sponginess of the gums, and a tendency to hemorrhages into subcutaneous tissues or from mucous membranes.

Etiology.—The disease appears at all ages and in both sexes. It is most often seen in infants fed on sterile artificial foods, or in adults living on a diet deficient in fresh vegetables and fresh meats. Sailors are therefore most often affected, but it is met with at times among the inmates of prisons or asylums or among the very poor. Any cause lowering the vitality predisposes to the disease, especially insanitary surroundings, hard work, mental anxiety, and the like. Rarely it develops in healthy individuals without apparent cause.

Theories of the Disease.—A number have been propounded.

(1) That it is due to deficiency of potassium salts in the food and so in the blood. Hence the importance of fresh vegetables which are rich in potash salts.

(2) That it is caused by diminished alkalinity of the blood, due to the withdrawal of alkaline salts from the food.

(3) That it is caused by ptomaines developed in canned foods and salted meats, the usual diet of those who suffer from scurvy.

(4) That it is a specific infection to which certain conditions, such as the diet and surroundings commonly associated with scurvy, predispose. At present no one theory has won general acceptance.

Morbid Anatomy.—Effusions of blood into the skin, or subcutaneous tissues, and beneath the bones are the only constant lesions. The gums are swollen, soft, and possibly ulcerated. Hemorrhages may also be found in the serous membranes and in the joints. The blood effusions may be circumscribed or diffuse. Separation of epiphyses may occur in the young. Various complicating lesions, such as pneumonia, pleurisy, or nephritis, may be present.

Symptoms.—Prodromal symptoms are usually observed for days or weeks before the appearance of the typical symptoms. The patients

become anemic, feeble, and mentally apathetic. Gradually certain typical signs appear.

(1) The gums become spongy, especially about the incisor teeth, and bleed easily. Ulceration may follow; the teeth may be loosened and fall out. A bloody discharge from the mouth and a fetid breath accompany these changes.

(2) Hemorrhages occur into the skin or subcutaneous tissue. These may have the usual character of petechiæ, or ecchymoses, or they may form large hematomata, over which the skin is reddened and edematous, and which are very painful. As these hematomata most often occur about joints, standing or walking becomes painful.

(3) Hemorrhages from mucous surfaces, especially the nose and mouth, occur.

(4) Anemia becomes marked and the patients suffer consequently from debility, dyspnea on exertion, palpitation of the heart, loss of appetite, and constipation.

(5) Dysentery not infrequently results from the diet and insanitary conditions, and there may be blood in the stools.

Course.—The disease, once established, continues until the condition is recognized and proper treatment instituted or till the death of the patient from exhaustion, or from complications, such as pneumonia or gangrene of the lung. The blood shows a secondary anemia with moderate leukocytosis.

Diagnosis.—The conditions under which the disease develops usually make its recognition easy. Sporadic cases give rise to difficulty. The history of long-continued use of canned or preserved foods, the affection of the gums and the hemorrhages are characteristic. The result of treatment is the final test.

Purpura or the cachexia of chronic mercurial poisoning must be excluded. In sporadic cases the disease is most often mistaken for some form of rheumatism.

Treatment.—Removal to better hygienic conditions and a diet of fresh vegetables are the essentials. Fresh meat and milk are also valuable. Fresh orange and lemon juice and infusions of malt are credited with special antiscorbutic power. The condition of the mouth must be treated by cleanliness and an antiseptic mouth wash of peroxide of hydrogen or permanganate of potash, or boric acid.

The prevention of scurvy lies in the maintenance of hygienic surroundings and a dietary containing sufficient vegetable food, especially fresh vegetables. Lime juice is given, 2 ounces per week.

INFANTILE SCURVY

While scurvy among the adult population has almost disappeared by reason of proper preventive measures, the disease has become not uncommon among infants because of the general adoption of artificial foods.

Etiology.—A very few cases of scurvy in breast-fed children are on

record.   Sterile artificial foods, such as the proprietary cereal foods, condensed milk, sterilized milk, and even Pasteurized milk are the common causes of scurvy in infants.   Scurvy develops only after the infant has been fed for some months, as a rule, on one of these substitutes for breast milk, and it is therefore most often seen during the second six months of life, and rarely during earlier months.

The cause of scurvy in these cases appears to be a deficiency of certain alkaline salts, such as citrate of lime, in the food, with resulting diminished alkalinity of the blood, although this reduction of the alkalinity of the blood in scurvy has not been proven.   The addition of these salts, in a fresh diet, or even in sterilized fruit juice, cures the disease.

Morbid Anatomy.—Subperiosteal hemorrhages are the characteristic features of scurvy in infants.   These hemorrhages are found on the long bones, just above the knee, at the ankle, wrist, or elbow.   The bones may be rarefied and epiphyses may be separated.   Hemorrhagic effusions in the skin, joints, or serous surfaces may also be found.   The gums may be swollen and spongy.

Symptoms.—The invasion is usually insidious.   The children become fretful and cry out, when handled.   They are restless and sleep poorly. The characteristic symptoms then appear.

(1) Pain on any motion of the legs, so severe that the child cries out when touched or even approached.   So carefully is voluntary motion avoided that the condition is sometimes mistaken for paralysis.   Rheumatism is the common diagnosis.

On examination one or both legs is found swollen above the knee or at the ankle.   The swellings are exquisitely tender.   The skin is often glossy, possibly edematous.   Instead of the legs, the arms, the ribs, or clavicle may be affected.

(2) The gums about the incisor teeth are swollen, deep blue, and soft (spongy) so that they bleed easily.   If no teeth are present the gums show little or no change.

(3) Hemorrhages into the skin from mucous membranes, especially hematuria, may occur.

(4) Fever of moderate degree may be present.

(5) Rickets and scurvy are often associated, because they are both related to deficiency of the diet.

Diagnosis.—Painful motion of the legs and spongy bleeding gums in an artificially fed child justify the diagnosis.   The affection is often miscalled rheumatism or mistaken for paralysis or rickets.   In case of doubt the test of a change of diet will promptly settle the question.

Prognosis.—If the disease is recognized and properly treated, recovery is wonderfully prompt and sure.   Death may occur from exhaustion, bronchopneumonia or diarrhea.

Treatment.—Sterilized or canned foods must be stopped and a proper dilution of fresh cow's milk or breast milk prescribed.   Orange juice sweetened with sugar, if necessary, should be given, one or two teaspoonfuls thrice daily.   The affected limbs should be kept as quiet as possible.

STATUS LYMPHATICUS

Definition.—A rare constitutional condition of children or young adults characterized anatomically by enlargement of the thymus and a hypertrophy of all the lymphoid tissues, and clinically by liability to sudden death from trivial causes.

Etiology.—The cause of the condition is unknown. It is closely associated with rickets and appears to be due to like causes, malnutrition and defective hygiene.

Morbid Anatomy.—(1) The lymph nodes throughout the body, especially the cervical, thoracic and abdominal, are markedly enlarged; the tonsils, the lymphatic tissues of the nasopharynx and root of the tongue, solitary and agminate follicles of the intestine are hypertrophied. (2) The spleen is moderately enlarged. (3) The thymus is enlarged and weighs much more than the normal organ (see Enlarged Thymus). (4) The bone-marrow shows a lymphoid hyperplasia. (5) The heart and aorta are often smaller than normal, the so-called hypoplasia of these structures.

Diagnosis.—The subjects of this disorder are usually well-nourished, fat but flabby children. The enlargement of tonsils, adenoids, and the superficial lymphatic glands can be made out. Dulness under the manubrium or skiagrams of the chest may show enlargement of the thymus and the mediastinal glands; the mesenteric glands may be palpable.

Relation to Sudden Death.—Certain cases of sudden death following the injection of diphtheria antitoxin, the inhalation of chloroform or ether, or trivial operations such as circumcision, have been found to be associated with the condition of status lymphaticus. Death has been ascribed in some cases to pressure of the enlarged thymus gland (see Enlarged Thymus), but evidences of pressure are often lacking and the exact cause of death in these cases is not established.

DISEASES OF THE SUPRARENAL BODIES

ADDISON'S DISEASE

Definition.—A constitutional disease characterized by asthenia, feeble circulation, gastric irritability and pigmentation of the skin.

Etiology.—The disease is rare. Men between twenty and forty years are most often affected. Although the lesion of the adrenals is most often tuberculous, the disease is exceedingly rarely accompanied by evidences of other tuberculous lesions.

Morbid Anatomy.—Tuberculosis of the adrenals, most often as a general caseation, sometimes as isolated tubercles or large tubercular tumors, is the most frequent lesion. Other lesions of the adrenals, cancer, sarcoma, gumma, etc., have also been found to cause the disease.

Involvement of the abdominal sympathetic has been observed and has been thought to account for certain cases in which the adrenals were normal, the nerve lesion resulting in inhibition of the function of an anatomically sound gland.

Pathogenesis.—It is now generally accepted that the disease is caused by an absence of an internal secretion of the adrenals, due to tuberculous or other lesions, or to functional inactivity caused by lesions of the sympathetic nervous system.

Symptoms.—The invasion is very insidious as a rule, the only impressive feature being increasing weakness.

(1) The physical weakness is associated with mental apathy. Body and mind weary without exertion and exhaustion is produced by any effort.

(2) Gastric irritability marked by loss of appetite, nausea and vomiting without sufficient cause exists throughout the disease. It shows itself markedly at times, with intervals when there is no disturbance.

(3) Pigmentation of the skin is quite constant, affecting usually the exposed parts, the hands and face and situations like the axillæ, abdomen, groins, or the genitals. The color of the skin varies from light yellow to black. It is usually progressive. Rarely it is lacking altogether. The buccal mucous membrane may show similar pigmented areas.

(4) The pulse is small and very feeble, with little increase in rate. The blood-pressure is low.

These symptoms constitute the essentials of the clinical picture.

Course.—Once the disease is established it usually goes on steadily to a fatal conclusion, caused by syncope, or simply increasing asthenia. The end in most cases comes within one year, but some cases last five or even ten years. Recovery has taken place.

Diagnosis.—The grouping of symptoms is characteristic, but the utmost care must be taken to exclude other possible causes of the asthenia, such as tuberculosis, cancer, myocarditis and the like.

Tuberculin reactions should be tested for by von Pirquet's method or by subcutaneous inoculation.

Treatment.—The administration of suprarenal extract should be tried, in tabloids of one grain, representing 15 grains of the gland, thrice daily. Unfortunately only a small part of the cases react favorably to this rational treatment. Otherwise treatment must be purely symptomatic, rest, food, fresh air, and tonics.

DISEASES OF THE SPLEEN

MOVABLE SPLEEN

Etiology.—Abnormal mobility of the spleen is almost always due to enlargement caused by previous malaria, syphilis, tuberculosis, leukemia, splenic anemia or other disease. In other cases it is associated with enteroptosis due to congenital defects of the ligaments or relaxation caused by subsequent strain.

Symptoms.—As a rule there are none. A large spleen, when displaced, may, however, cause symptoms by pressure. Pain or dragging may be complained of. Torsion of the pedicle may cause acute necrosis

with intense pain, nausea, vomiting and prostration. Pressure upon the uterus may cause metrorrhagia.

Diagnosis.—This must rest on the recognition of the displaced viscus, by its smooth external surface, general shape, and sharp, notched anterior border.

Treatment.—An abdominal belt with support for the spleen may be effective. Opening the abdomen and packing the organ in position till adhesions have formed have also been successful. Removal has been done when other measures failed.

RUPTURE OF THE SPLEEN

Etiology.—Rupture occurs spontaneously or as the result of a fall or blow, most frequently of spleens enlarged for some reason, rarely in the normal organ. The symptoms are those of internal hemorrhage, severe pain, pallor, dyspnea or air-hunger and collapse with small, weak pulse. Immediate operation is demanded and usually the removal of the spleen.

INFARCT AND ABSCESS OF THE SPLEEN

Infarcts of the spleen are produced by the plugging of one of the branches of the splenic artery by emboli. These emboli usually come from the vegetations of endocarditis or the softened thrombi of pyemia or other acute infections, such as typhoid fever. The infarcts appear as pyramidal areas, deep red and firm at first, later white and fibrous. If produced by septic emboli they may soften or break down and form abscesses. The indications of infarction are pain, referred to the splenic region, tenderness and swelling of the spleen. A friction rub may be present from accompanying perisplenitis. Abscesses give the general symptoms of septicemia with local pain, tenderness, and enlargement of the organ. Peritonitis may be caused by extension or by rupture of the abscess.

TUMORS OF THE SPLEEN

Tubercles and gummata are often found post-mortem, but are rarely recognized clinically, owing to the greater prominence of lesions in other organs. Sarcoma and carcinoma as primary growths are exceedingly rare. Echinococcus cysts are also known.

Amyloid spleen is present as part of an amyloid degeneration affecting also the liver and kidneys. The spleen is sometimes enormously enlarged.

SPLENOMEGALY

(Splenic Anemia. Banti's Disease)

Under this heading are grouped several different affections characterized by marked, sometimes enormous enlargement of the spleen, and a secondary anemia. Several types may be distinguished (Osler).

(1) Simple splenomegaly, in which the spleen is greatly enlarged with little or no anemia and only slight dragging or pain from the weight of the organ (see Fig. 49).

(2) Splenomegaly with marked secondary anemia, a tendency to hemorrhages, and pigmentation of the skin. The hemorrhages occur from various mucous membranes, especially the stomach. Cirrhosis of the liver with ascites may develop in the final stages (Banti's disease). These conditions persist for many years.

(3) A family or infantile type, which begins in childhood and in many cases affects several members of a family. Cirrhosis of the liver, jaundice, pigmentation of the skin and stunting of the growth of the
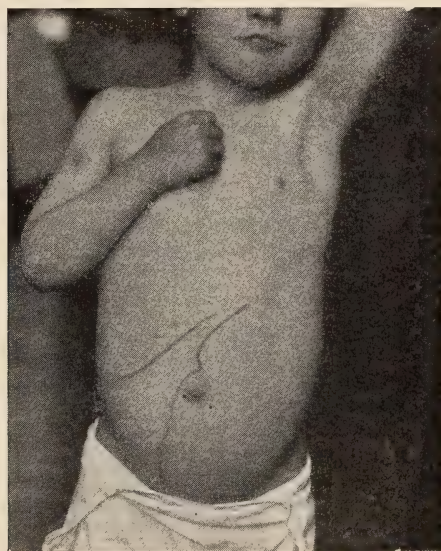
Fig. 49.– Splenic anemia: the lines mark the edges of liver and spleen.

children have been observed. In some cases a simple fibrosis of the spleen is present, in others a very remarkable endothelial hyperplasia involving not only the spleen, but the liver, lymph nodes and the medulla of the bones. Various names have been given to the condition, endothelioma, chronic endothelial hyperplasia, Gaucher's type of splenomegaly, etc.

The diagnosis of these conditions rests upon the associated enlargement of the spleen and anemia, in the absence of any of the usual causes for chronic splenic enlargement, such as rickets, syphilis, tuberculosis, malaria, leukemia, Hodgkin's disease, or chronic polycythemia, etc.

Treatment of the anemia and exposure to the X-rays have given some relief. Splenectomy has been successful in a few cases.

POLYCYTHEMIA
(Erythrocytosis Megalosplenica)

Definition.—A rare disease characterized by cyanosis, increase in the number of red corpuscles per cubic millimeter of blood and enlargement of the spleen.
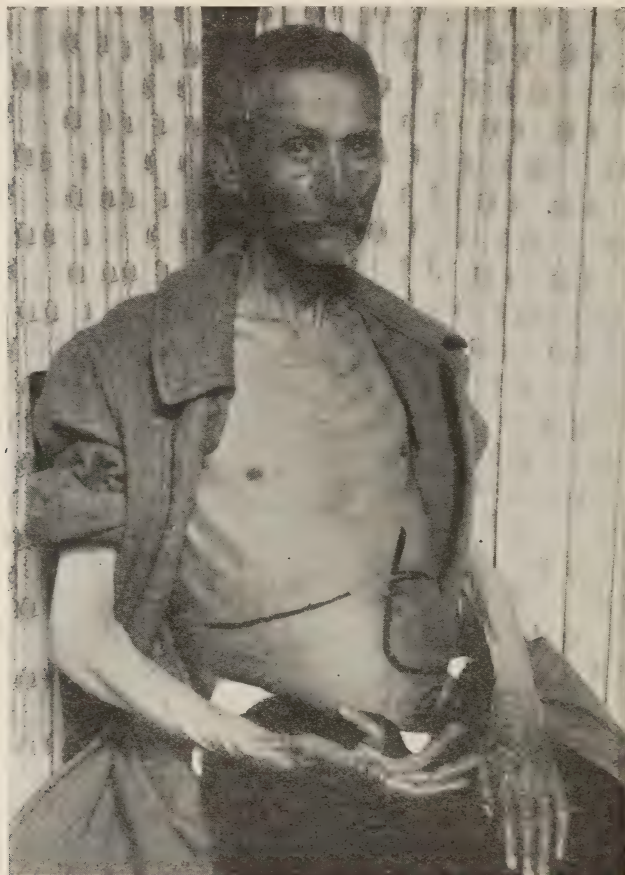
Fig. 50.—Polycythemia with cyanosis (of the face and hands). The margins of enlarged spleen and liver marked on the abdomen. From the collection of Dr. A. R. James.

Etiology.—The cases thus far reported include both men and women from 35 to 60 years of age.

Symptoms.—The patients complain of headache, vertigo or weakness. Sometimes the color of the skin and mucous membranes excites concern. This color is a deep bluish-red, the lips and finger-nails look blue, as in cyanosis (see Fig. 50). Hemorrhages from the mucous membranes, neuralgia, dyspnea and edema of the lungs have been observed. Temporary improvement may occur, but the disease persists indefinitely.

Examination shows an enlarged, often an enormous, spleen and the polycythemia. The red cells exceed 8,000,000 and may number 13,000,000 per c.mm. The hemoglobin varies from 120 to 190 per cent., and the leukocytes are increased, with a relative increase of the polynuclears, even to 92 per cent.

Treatment.—Bleeding gives temporary relief. X-ray treatment reduces the spleen and the number of red cells.

DISEASES OF THE THYROID GLAND

CONGESTION

Congestion of the thyroid is especially associated with changes in the menstrual function, so that it appears at puberty, with the menstrual period and, it is said, with defloration. It may also be produced by tight collars, prolonged crying, epileptic fits and the like. Beyond the swelling no symptoms are produced and the condition is transient.

ACUTE THYROIDITIS

Acute inflammation of the thyroid occurs as (1) the result of the action of certain poisons, such as alcohol, iodine or phosphorus, or (2) as a complication of acute infectious diseases, such as typhoid and scarlet fever, measles, small-pox, etc. The inflammatory process may be simple or suppurative. In the suppurative cases various organisms, staphylococci, streptococci, the typhoid bacillus, etc., have been recovered.

Symptoms.—The gland is swollen, and in the severer cases tender. Usually one lobe is the more affected. In suppurative cases, the skin becomes reddened and fluctuation develops. The enlarged gland may give the usual signs of pressure, congestion of the neck and face, dyspnea or dysphagia. Graves' disease or myxedema may follow.

Treatment.—A light ice-bag may be applied and rest enjoined. Suppurative conditions must be promptly opened.

GOITRE

Definition.—A chronic hypertrophy of the thyroid gland.

Etiology.—The affection occurs sporadically and endemically. Sporadic cases occur in women in all parts of this country. The disease is endemic in certain mountainous regions, especially Switzerland and parts of France, also in South America and Asia. In Switzerland especially goitre is frequently associated with cretinism. The drinking water is usually regarded as the cause of the affection.

In North America endemic centers have been found in regions at the eastern end of Lake Ontario, in Michigan and the mountainous parts of Pennsylvania. Epidemics have been observed in schools and garrisons.

Morbid Anatomy.—In general there is an increase in all parts of the gland. Certain varieties are named from the predominant features of the process. (1) Parenchymatous, when the glandular structure is most hypertrophied, the enlarged follicles being filled with normal colloid material. (2) Cystic, when the follicles are so much enlarged as to give the impression of cysts. These cysts may be formed by enlargement of a single follicle or the fusion of several. (3) Vascular, when the vessels are notably dilated. The enlarged vessels frequently show atheromatous changes. (4) Fibrous, when the interstitial tissue is greatly increased and the gland is firm or hard.

Symptoms.—The whole gland may be uniformly enlarged or only one lobe, especially the right. The enlargement may be slight or extreme in degree. Symptoms consist only of the appearance of the tumor and pressure if it be large. Dyspnea from pressure on the trachea, rarely dysphagia from obstruction of the esophagus, congestion of the head and neck from pressure on veins, and disturbance of the heart from pressure on the pneumogastric nerves may result.

The growth is painless, and is soft, cystic, or hard, according to the degree of involvement of the several parts of the gland. Pulsation may be felt and a systolic murmur heard in cases of vascular goitre.

Diagnosis.—Goitre is distinguished by the characters of the tumor and the absence of the symptoms observed in Graves' disease. The tumor moves upward in deglutition.

Prognosis is good as to life. Sudden death may occur from tracheal stenosis, disturbance of the heart, edema of the glottis or apoplexy, but is rare.

Treatment.—Removal from a goitrous district and the boiling of drinking water are employed as prophylactic measures. Iodine is given either as potassium iodide, 5 to 10 grains thrice daily, or 1 dram of the liquor iodi compositus. Excessive dosage may cause symptoms resembling Graves' disease, nervousness, palpitation, sweating, tremor, etc. X-ray treatment has also been effective. Surgical measures may be required for relief of pressure.

TUMORS OF THE THYROID

Adenoma, simple or malignant, sarcoma and cancer are known, but are very rare in comparison with the common goitre. These tumors have their usual characters. Symptoms are due chiefly to pressure. Treatment is surgical.

EXOPHTHALMIC GOITRE
(Graves' Disease. Basedow's Disease)

Definition.—A disease due to morbid function of the thyroid and characterized by exophthalmos, goitre, tachycardia, and tremor, or by any combination of these cardinal symptoms.

Etiology.—Women are more affected in the proportion of 6 or 8

to 1. The disease usually appears between the ages of 16 and 40. Beyond these facts little of value is known concerning the causation of the disease.

Fright or protracted worry is found to precede the onset in some cases, in others some acute infection, such as tonsillitis, and in still others intestinal disturbances, but the influence of these factors is not marked.

Moebius' view that exophthalmic goitre is an intoxication due to morbid function of the thyroid gland is now generally accepted. The antithesis to myxedema presented by exophthalmic goitre is striking. The results of the administration of excessive doses of thyroid gland are also very suggestive of exophthalmic goitre. Even the exophthalmos has been produced in this way. It is, however, clear that exophthalmic

FIG .51.—Exophthalmic goitre: showing the exophthalmos and the goitre.

goitre is due not alone to excessive functioning of the gland, but also to abnormal secretion.

Morbid Anatomy.—The thyroid gland is usually enlarged, but may be smaller than normal. The gland shows a diffuse fibrosis, with marked changes in the epithelium of the alveoli. The colloid is diminished or increased in amount and altered in quality. Whether these changes are peculiar to exophthalmic goitre is an open question.

The thymus is often persistent and enlarged. There are no other constant lesions.

Symptoms.—(1) Goitre of some kind is present in nearly all cases, but may be entirely lacking. The enlargement of the gland may be of any degree or type. Thus the gland is but slightly enlarged in some cases, quite large in others. The whole gland may be affected or only one lobe. The goitre may be soft and elastic or cystic, but is more often firm or hard. A thrill and a systolic, a diastolic, or a continuous murmur may be heard over the gland (see Fig. 51).

(2) Tachycardia with other symptoms of vascular excitation is usually present. The pulse rate varies from normal to 200 or more. Palpitation and dyspnea on exertion usually accompany the tachycardia.

The patients may also complain of the throbbing of the arteries in various parts of the body. Late in the disease the pulse often becomes slow and intermittent.

The heart is slightly enlarged, its impulse very forcible, and the shock of the heart sounds palpable. The throbbing of the carotids, the abdominal aorta and even the peripheral vessels is marked. The capil-

Fig. 52.—Graves' disease, showing the emaciation, exophthalmos, and goitre.

lary pulse or throbbing in the veins of the hand may be observed. Flushing of the face or other parts of the body is common.

On auscultation cardiac murmurs are usually heard, systolic murmurs at both base and apex, and loud, sharp heart sounds. The pulse often has the Corrigan quality.

(3) Exophthalmos—protrusion of the eyes—is usually present. The causes of this symptom are still in doubt. Congestion of the orbital vessels, increase of orbital fat, edema, and spasm of the special orbital muscle of Mueller have all been suggested. The ocular symptoms of the disease have been given the names of different observers.

The staring with diminished frequency of winking is known as Stellwag's sign.

Graefe's sign consists in failure of the upper lid to follow smoothly

a downward motion of the eyeball.   The lid lags behind or even jerks backward.

Moebius' Sign.—In near convergence of the eyes, as in fixing the eyes on the tip of the nose, one eye will turn outward, owing to muscular weakness.

Because of the diminished motion of the lids and loss of sensitiveness in the cornea, conjunctivitis or ulceration of the cornea may result in advanced cases.

(4) Tremor.—A rapid fine tremor is present in the fingers and possibly the tongue.   Associated with this tremor are various nervous symptoms, including mental excitability, irritability or restlessness. Mental depression or exaltation may occur.

(5) Various other symptoms are more or less commonly met with. Emaciation is frequent (see Fig. 52), especially late in the disease, and persistent diarrhea is common.   Fever is present in many cases, very irregular in its daily and weekly variations.

Course.—The evolution of the symptoms may be rapid or slow. Once the disease is established the course is chronic, usually lasting for years.

Recovery is not uncommon under treatment, either medical or surgical.   Death may result, either from the disease itself or complications, such as pneumonia or tuberculosis.

Diagnosis.—Fully developed cases can hardly be mistaken.   With one or more of the cardinal symptoms missing, doubts arise and opinions differ.

Treatment.—(1) Symptomatic.—Rest, both physical and mental, is necessary from the outset.   Many remedies have been advised and used. Digitalis or strophanthus to slow the pulse, belladonna and ergot to control the vascular disturbances, phosphate of soda and intestinal antiseptics to meet the assumed intestinal autointoxication.

(2) Organotherapy.—Various preparations made from the blood, the serum, or the milk of thyroidectomized sheep or goats are in use. The serum of rabbits treated by injection of the nucleo-proteid or thyroglobulin of the thyroids obtained from cases of Graves' disease has also been used with some success.

(3) Electricity and the X-rays have relieved or cured some cases.

(4) Surgical treatment is increasingly successful and should be advised after reasonable trial of other methods.   The ligation of the superior thyroid arteries or removal of part of the gland is usually recommended.

MYXEDEMA

Definition.—A disease caused by loss of function of the thyroid gland, and characterized by atrophy of the gland, mental hebetude, and a myxedematous condition of the subcutaneous tissues.

The condition may be due to congenital defect of the gland (cretinism), or to loss of function in adult life (myxedema) or the operative removal of the gland (operative myxedema).

CRETINISM

Etiology.—Cretinism occurs in both endemic and sporadic types. The endemic type is common in the mountainous regions of Switzerland, France and Italy. The sporadic cases are found under any conditions. Since the recognition of the disease many cases have been discovered in America.

Symptoms.—During the second six months of the child's life it is noted that dentition is delayed and that the child does not develop mentally. During the second year the typical features of cretinism appear. The form is stunted, the limbs short and thick. The hair is thin and dry. The tongue is thick and often protrudes from the mouth.

Fig. 53.—A cretin aged 25 years. Note the shrunken figure, expressionless face, protuberant abdomen with umbilical hernia, and kyphosis.
Fig. 54.—The same cretin, dorsal view, showing the lateral curvature of the spine.
From the collection of Dr. W. P. Northrup.

The face is expressionless, the eyes dull, the skin thick. The teeth, if present, decay early (see Figs. 53 and 54). Over the clavicles there are pads of myxedematous tissue. The body is heavy, the abdomen prominent, and umbilical hernia is common. The muscles are weak. The child walks late, talks late, and remains mentally undeveloped, an idiot or imbecile in many cases. The temperature is subnormal, the pulse slow. Unless relieved by treatment these characters persist into adult life, and from time to time typical cretins of 30 or 35 years are discovered.

MYXEDEMA

Etiology.—Myxedema affects women in the ratio of 6 to 1. Goitre or Graves' disease may precede it. The tendency to myxedema is transmitted in some families.

The symptoms are very like those of cretinism. Increasing mental

dulness is usually first noted, then the hair becomes thin and dry, the skin pale, sallow, dry and thick, especially in the face and extremities,
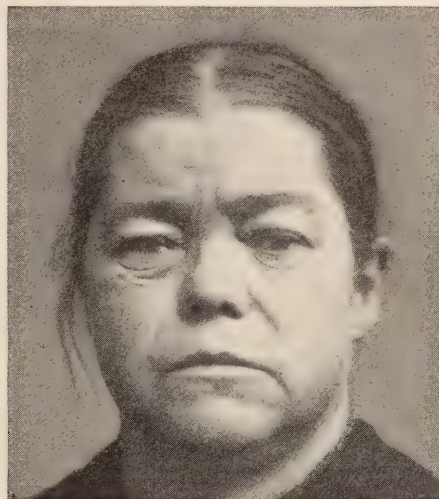
Fig. 55.—Myxedema, showing the thin, dry hair, the puffy eyelids, and the dull, heavy expression of the face.

so that the patients are often thought to have nephritis. The tongue grows thick, the speech slow and scanning and very limited. The nose

Fig. 56.—Myxedema. The backs of the hands are puffy, the skin dry, wrinkled, parchment-like.

becomes broad and thick (see Figs. 55 and 56). The bodily movements become slow and heavy. Memory fails and often the patients become

18

irritable or suspicious. Dementia may result. The temperature may be below normal and the pulse slow.

Operative myxedema or cachexia strumipriva was not uncommon following complete removal of the thyroid gland for goitre or other cause, but now is rarely seen since only partial removals are attempted. The symptoms are those of myxedema.

Diagnosis.—The diagnosis of cretinism is extremely easy, if one has the condition in mind. No other congenital defect closely resembles it. In mild cases doubt may arise. Treatment may then be tried experimentally.

Myxedema has often been mistaken for Bright's disease because of the swollen face and extremities and the presence of albumin and casts in the urine, a common finding. The mental dulness, the condition of the hair, and other features named should distinguish it.

Treatment.—The thyroid gland of the sheep is administered in tablets. For a child, 1/5 to 1/4 grain may be given thrice daily, for an adult 1 grain. The dose is then increased gradually to 10 or 12 times these amounts. Treatment may be stopped from time to time and renewed again. The results are often marvelous. In rare cases the treatment has no effect. The administration of too large doses of the thyroid gland produces fever, restlessness, rapid pulse, and prostration.

DISEASES OF THE THYMUS GLAND

Interest centers in the persistence and hypertrophy of the gland. The old teaching that the gland normally atrophies after the second year is incorrect. A table, prepared by Nicoll and the writer, of the weights of 495 glands from children varying in age from birth to five years showed the weight of the gland to average practically the same throughout that period. Cunningham, of Edinburgh, states that the gland regularly persists, but is lost to observation in the increasing mediastinal fat. Under certain conditions, however, the gland is found much increased in size.

Hypertrophy of the thymus is associated, as a rule, with a general hypertrophy of the lymphoid tissues (see Status Lymphaticus). Occasionally the hypertrophy of the gland is so great as to dominate the picture. Ever since the appearance of Friedleben's great monograph on the Thymus in 1858, discussion has been carried on regarding the possible relation of enlargement of the thymus to laryngismus stridulus or other dyspnea produced by pressure on the trachea, the so-called thymic asthma. Friedleben denied the influence of the thymus, others have affirmed it and the discussion is still open. The relation of enlargement of the thymus to sudden death is also still under discussion. (See p. 262.)

VI

CONSTITUTIONAL DISEASES

CHRONIC POLYARTHRITIS

Definition.—A constitutional disease characterized by chronic inflammation of a number of joints, anemia and malnutrition, the etiology of which is unknown.

Chronic polyarthritis may arise from a number of causes, some undoubtedly bacterial, as gonorrheal, tubercular, pneumococcic, or septic infections; some metabolic, such as gout and the purpuric affections; some of nervous origin, such as those occurring in syringomyelia, and locomotor ataxia. When all these groups have been separated off, there remains a considerable number of cases of chronic polyarthritis for which no satisfactory explanation can at present be given. For some years efforts have been made to classify these cases, as hypertrophic or atrophic or periarticular, according to the nature of the changes observed in the affected joints. No good result has been reached in this way, and the procedure is no more satisfactory than would be the division of tubercular arthritis on a like basis. It therefore seems best at this time to include all these cases under one broad term, substituted for the designation of Arthritis Deformans, which is applicable only to one of the several groups into which these cases readily fall when classified on anatomical grounds. It will be noted that chronic rheumatism is included. The term rheumatism in this connection no longer has any clear meaning and should be dropped.

Etiology.—The conditions under which chronic polyarthritis is met with may be stated, although they throw practically no light on the real cause of the disease. Children are but rarely affected. Adults of all ages and both sexes suffer from it. Elaborate studies of the age, race, sex, social condition, employment, and previous illnesses of patients have been productive of nothing of value. The metabolism of the patients has been studied, especially the nitrogenous metabolism, without result. In some studies the calcium and magnesium metabolism has been investigated, with the result that some cases show retention, others loss of these substances so closely related to bone formation. As stated in the definition the etiology of these cases is unknown.

Morbid Anatomy.—There is a chronic inflammation of a number of joints, usually symmetrically placed, and either large, such as the ankle, knee, or hip, or small, such as the finger-joints. The affected joints are large, either partly or completely ankylosed, the tissues about them are dense. The muscles of the part are atrophied, both above and below the joint. The skin is often dry and glossy and cracks easily. If the joints are opened, the secretion is found either increased or diminished. The synovial membranes are thickened. The cartilages are

fibrillated and often eroded. The capsule is thickened and dense. The articular ends of the bones are thickened, or atrophied, or in some cases exposed in the joint and eburnated. The periarticular tissues are much thickened and adherent, tendons about the joint being firmly bound down.

According to the degree in which the several parts of the joints are affected in different cases the lesions have been classified as (1) Hypertrophic—in which new bone has been formed about the joints, producing the so-called osteophytes. As a result of new formation of bone at one point and erosion at another the joints are greatly deformed (arthritis deformans). (2) Atrophic—in which there is rarefaction of the articular ends of the bones forming the joints and atrophy of other tissues, so that the joints may seem smaller than normal. (3) Periarticular—the chief lesion being found about rather than in the joint, the periarticular tissues being swollen, thickened, and densely adherent. This is the most common form and is usually denominated rheumatoid arthritis.

Symptoms.—1. MONARTICULAR TYPE.—A number of joints are usually affected in some degree, but the process becomes so marked in a single large joint, the hip, knee, or shoulder, that it dominates the picture. The joint is painful, especially on motion, enlarged and deformed, and motion becomes progressively restricted. The muscles above and below the joint atrophy. From time to time the pain and restriction of motion are aggravated. The condition is very chronic and may result in complete ankylosis (see Fig. 40). 2. VERTEBRAL TYPE: The joints of the spinal column are involved, sometimes in part, sometimes throughout. Pain and limitation of motion are the marked features, the spine becoming more or less rigid (poker back). Pain is felt in the back or, if intercostal nerves are involved, referred to the chest or abdomen. As in the other forms, the disease is chronic and progressive and the disability resulting may be severe. The general health may suffer from the constant pain. 3. HEBERDEN'S NODES: The joints of the fingers, especially the terminal phalanges, are involved in this type, often without participation of other joints. The articular ends of the bones are roughly enlarged by exostoses, the joints suffer to some extent, with pain and limitation of motion, and sometimes marked deformity from deflection of the terminal phalanges. Exacerbations occur as in other types. Often no other symptoms are complained of and the patients think little of the slight deformity and disability. 4. GENERAL PROGRESSIVE TYPE: An acute and chronic form are described. (a) The acute type closely resembles the milder attacks of rheumatic fever. A number of joints are swollen, tender, and painful, and the patients suffer from fever and become anemic as in rheumatic fever. These attacks are regularly mistaken for rheumatic fever. The joint lesions are not so red, or so exquisitely tender as in the later affection, they do not yield to the salicylates, but persist and develop characteristic lesions of the bones and joints. (b) Chronic type. In this form one

joint after another is involved by a chronic progressive arthritis which produces swelling, pain, and limitation of motion (see Fig. 57). The disease may progress from one joint to another of the body till all are ankylosed to a greater or less degree (see Fig. 58). From time to time the joint symptoms are intensified, and the temperature is raised as in an acute attack. The patients suffer from impaired digestion and become anemic, and the muscular atrophy about the affected joints may be extreme. In its severe form the disease reduces the patient to a helpless invalidism. The blood shows the changes of a secondary anemia.

Diagnosis.—The diagnosis of chronic polyarthritis or arthritis deformans implies the exclusion of all the known causes of such con-

Fig. 57.—Chronic polyarthritis. Arthritis deformans: showing the enlargement and deformity of the joints of the wrists and fingers.

ditions. Rheumatic fever can easily be confused with the first acute attacks of the general progressive type. Failure of the salicylates to produce any effect on the process is suggestive. The cardiac complications, involvement of the pleura and pericardium belong to acute rheumatic fever, and not to chronic polyarthritis. The after course of the affection makes the differentiation easy, as chronic polyarthritis tends to persist and produces progressive changes. Gonorrheal arthritis must be excluded by a knowledge of the history, examination of the urethra or vagina for gonococci, or the fluid of joints, if effusions are present. Sometimes a therapeutic test of the influence of gonococcus serum or vaccines is indicated. Tuberculosis must be excluded by a study of the lesions, the absence of any tendency to the formation of

pus or discharging sinuses in chronic polyarthritis, and in some cases by von Pirquet's test or the subcutaneous inoculation of tuberculin. Septic arthritis can be excluded only by the clinical history, the absence of any preceding disease such as scarlet fever, pneumonia, or endometritis. Skiagraphs constitute a most valuable aid in determining the nature and extent of the changes in and about the joints.

Prognosis.—This varies to some extent in the different types of the disease. The monarticular type tends to disablement of the joint most involved, but the general health suffers little. Heberden's nodes, as a rule, affect the life and comfort of the patient hardly at all. The progressive type is severe and, while the disease is not fatal, tends to

Fig. 58.—Case of arthritis deformans from Dr. Young's clinic. (International Clinics, Vol. III, Series 20.).

permanently cripple the patient and possibly renders him quite helpless. Death results from intercurrent disease.

Treatment.—Medicines have no specific effect upon the condition. Treatment must be hygienic and dietetic. Residence in a dry, mild climate is desirable. The diet must be suited to the digestive capacity of the patient and must be as nutritious as possible. Restriction of proteid food such as is practiced in gout is not beneficial. Abundance of fat in the form of butter and cream is indicated. The patients must be encouraged to take such exercise as is possible without severe pain. Continued inactivity tends to increase the disability. For the arthritis rest and immobilization should be practiced only in the acute exacerbations and then for a limited time. Hydrotherapy is employed in various forms. Alternate applications of hot and cold water, or hot and cold douches, to the affected joints, are often beneficial. The hot-air treat-

ment, baking the joints for 10 to 15 minutes in dry air at a temperature of 250° to 300° or even more, is often employed. Electric-light baths, either local or general, have a similar effect. Massage with passive movements of the joints is helpful in preventing muscular atrophy and maintaining the motion of affected joints.

MUSCULAR RHEUMATISM
(Myalgia)

Definition.—An affection characterized by pain in certain groups of muscles. In its literal meaning the term is often used to cover any muscle pain. Clinically its application is limited to pains in muscles not accounted for by neuritis, by the presence of parasites (trichinæ), or by trauma, or tumor or other definite lesion. According to the muscles involved the disease is termed torticollis (muscles of the neck), pleurodynia (the intercostals), lumbago (muscles of the back), scapulodynia (muscles of the shoulder), etc.

Etiology.—The causes commonly include: (1) Exposure to wet or cold. (2) Trauma. (3) A rheumatic or gouty diathesis.

Morbid Anatomy.—The affection was long regarded as a neuralgia without definite anatomic lesions. Later it was supposed to be a myositis. Recent studies have shown the only definite lesions to be in the white fibrous tissue sheathing the muscles and nerves, and forming tendons and periosteum. The lesions found were swelling, edema and degeneration of the fibrous tissue.

Symptoms.—Pain is the one constant symptom. It may be acute and sharp or a dull aching. It is regularly aggravated by motion and relieved by rest. Usually it comes and goes in varying severity. An attack usually lasts from a day or two to a week. In some cases the condition becomes chronic. Recurrences are frequent. Torticollis derives its name from the fact that with the pain the head is turned to one side by contraction of the affected muscles. Lumbago may also show contraction of the muscles involved. On examination the muscles may be found tender to pressure, motion aggravates the pain, and in some cases definite nodules may be found in the muscles. These nodules are small indurated areas, feeling like shot imbedded in the tissues.

Treatment.—Rest for the affected muscles is of first importance. This is secured by strapping the lumbar or intercostal regions, or by rest in bed in severe cases. Counter-irritation with the Pacquelin cautery is often helpful. Heat in other forms (the hot water bottle or electric light) may be employed. Acupuncture has been successful in lumbago. Of medicines sodium salicylate, gr. x–xv, every 3 or 4 hours, or aspirin, gr. x, may be given. Colchicum has better effects in some cases. The chronic or recurrent cases are treated for a gouty diathesis by reduction of proteid food, the free drinking of water, regulated exercise, and hydrotherapy, hot baths and packs.

GOUT

(Podagra)

Definition.—A disorder produced by defective nitrogen metabolism and characterized by excess of uric acid in the blood and recurrent attacks of arthritis due to the deposition of sodium biurate.

Etiology.—A number of factors are active in most cases, such as (1) Heredity. The disease or tendency to it may be marked in successive generations of a family, doubtless from like habits of living. (2) The excessive use of alcohol, especially in the fermented beverages, such as wines, beer and ale. The distilled liquors, whiskey, rum, and gin, appear less harmful. (3) Overfeeding, especially if the diet be over-rich in meat or other nitrogenous foods. (4) Deficient exercise. Lack of proper relation between food and exercise is the more accurate explanation of overfeeding. (5) Lead-poisoning is, in England, a frequent antecedent. Apparently it is less frequently seen in this country.

Morbid Anatomy.—THE BLOOD. An excess of uric acid may be demonstrated by Garrod's thread test. To 2 drams of serum, taken either from the drawn blood or from a blister, 6 minims of moderately strong acetic acid are added in a flat water crystal. A fiber or two of linen thread is then introduced and the preparation set aside for from 36 to 60 hours. If uric acid is present in excess it will be found in crystals upon the thread. The test often fails.

THE JOINTS.—The affected joints are enlarged, the periarticular tissues thickened, and deposits of sodium biurate in the form of tophi are present in the cartilages of the joints and in the periarticular tissues. The tophi sometimes form masses of considerable size about the joints, or there may be only small nodules. When opened, the tophi are found to contain a thick, paste-like material, which has been shown to be mainly sodium biurate. The cartilages of the involved joints may be eroded in advanced cases. Tophi or chalk-stones are also found in the lobes of the ears, in the tendons and subcutaneous tissues, rarely in the laryngeal cartilages or cords and other sites.

The ASSOCIATED LESIONS of gout are found in the kidneys, heart, and arteries. The kidneys show the lesions of chronic interstitial nephritis, commonly in the form of contracted kidney, in other cases the larger, hard, red kidney of arteriosclerosis. The arteries, both the aorta and the peripheral vessels, present a marked condition of arteriosclerosis. The heart shows hypertrophy of the left ventricle, frequently accompanied by sclerosis of the coronary arteries and myocarditis or fatty degeneration of the heart muscle. Pericarditis may be found.

Chemistry of Gout.—As the result of clinical study and experimental investigation, the following propositions may be said to be fairly established. Gout is due to a disturbance of the nitrogenous metabolism of the body. The blood in gout is found to contain an excess of uric acid. This uric acid is derived mainly from the breaking down of nucleoproteids of the cells either of the body itself or of the food, and accord-

ing to its source the uric acid is therefore designated as endogenous or exogenous. A part of the endogenous uric acid has been shown to be a product of muscle metabolism. According to the generally accepted theory of Emil Fischer the nuclein or nucleic acid of cell nuclei contains a hypothetical base called by him purin. Uric acid and the so-called xanthin or purin bases (xanthin, hypoxanthin, guanin and adenin) are all derived from this hypothetical substance by a process of oxidation. This oxidation is brought about not by the simple step of chemical combination, but through the intervention of certain ferments. These ferments are not uniformly present in all the fluids or tissues of the body, but are found especially in the pancreas, adrenals, thymus, and spleen. Similarly the final step in the transformation of uric acid into urea is brought about by a special ferment shown to be present in the liver, muscles, kidneys, and bone-marrow. In the light of present evidence it seems probable that the defective nitrogenous metabolism characteristic of gout is brought about either by the absence or inhibition of this ferment called oxidase, which has the power of oxidizing and so converting uric acid into urea and other products.

Symptoms.—Acute Gout.—The onset is sudden with severe pain in one joint, usually the metatarso-phalangeal of one or the other great toe. The joint is swollen, red and very tender. The temperature rises to 101° or 103°. The patient's suffering is intense. With the morning the pain and fever subside, usually to recur the following evening for a week or ten days. The inflammation then subsides and the patient is well for an indefinite time; sooner or later the attack is repeated. Other joints than that of the great toe, especially the tarsal, may be first affected. Chronic Gout.—With repeated attacks other joints become involved, the tarsus, ankle, knee, wrist, or elbow, rarely the hip, or shoulder. Recurrences, much like acute attacks, become frequent. The affected joints become thickened, stiff, and studded with tophi of varying size. The tophi sometimes form tumors of considerable size, and may ulcerate and discharge their thick, paste-like contents, which on microscopic examination show crystals of sodium biurate. Tophi may also appear in the cartilages of the ears or in tendons, the subcutaneous tissues, or even in the larynx, etc. Gradually the arteries thicken, the heart hypertrophies, and the urine presents the changes of chronic interstitial nephritis. The course of the disease is exceedingly chronic, the attacks of arthritis being separated by long intervals of comparative comfort or illness due to some of the complicating conditions. The patients are also likely to suffer from chronic bronchitis and emphysema. The fatal termination is usually brought about by an attack of apoplexy, or uremia, or dilatation of the heart due to the myocarditis or to an intercurrent acute pericarditis.

Irregular Gout. Lithemic or Uric Acid Diatheses.—Gouty Diathesis.—Under this title are grouped a great number of minor disturbances of one or another tissue or organ of the body believed to be due to the same defective nitrogenous metabolism which underlies gout. These disturbances may affect any part of the body, the eye, the throat, the

heart, lungs, kidneys, skin, etc. They are met with (1) in persons who suffer from typical gout; (2) in individual members of gouty families, who are free from other evidences of the disease; (3) in certain people who have never had gout and do not belong to gouty families, but who have indulged excessively in food and drink. The evidence of the relationship in these latter cases depends wholly upon the curative influence of treatment along the lines of gout, and not upon the chance finding of uric acid crystals or urates in the urine. There is no question that the terms gouty or uric acid diathesis have been used for years very loosely to cover many indefinite affections whose only claim to the designation lay in the fact that the physician knew no other titles to give them and sought support for his diagnosis in the demonstration of deposits of uric acid or urates in the urine. On the other hand, there is no doubt that the defect in nitrogenous metabolism characteristic of gout is capable of producing a great variety of disturbances in different parts of the body. The more common are found in (1) the skin. Attacks of eczema, psoriasis, or herpes zoster are met with. Eczema of the face, ears, or neck is the most frequent. (2) The eye. Conjunctivitis and iritis are in some cases of gouty origin. (3) The Throat.—Attacks of pharyngitis with much congestion of the tissues are not common. (4) The Lungs.—Chronic bronchitis or emphysema, with asthmatic paroxysms, may occur. (5) The Heart.—Rapid or irregular action of the heart associated with attacks of pain resembling angina pectoris is described. (6) The Alimentary Tract.—So-called bilious attacks, marked by constipation, coating of the tongue, headache, and prostration, may be due to this cause. (7) The Urinary Tract.—Renal calculi are common in the gouty. Pyelitis, cystitis, or even urethritis may be due to the gouty diathesis. (8) The Nervous System.—Headaches, migraine, sciatica, neuralgia, cramps in the muscles of the leg and abdomen are found at times to be due to a gouty diathesis.

RETROCEDENT OR SUPPRESSED GOUT.—In the course of an acute attack, subsidence or improvement in the local conditions may be marked by severe internal symptoms, referred especially to the stomach or heart. The patient may suffer from abdominal pain, vomiting, diarrhea and profound depression and may die in the attack. In other cases the pain is referred to the heart and the pulse is rapid or slow and irregular. Acute pericarditis sometimes develops under these conditions.

Urine in Gout.—An immense amount of study has been devoted to the urine in gout, with but small results. A trace of albumin and a few hyaline or granular casts are usually found. A trace of sugar is not infrequent. In chronic gout the urine shows the evidences of chronic interstitial nephritis, is increased in amount, pale and of low specific gravity, with a trace of albumin and hyaline or granular casts. The excretion of uric acid varies greatly. Before an acute attack for two or three days the excretion is diminished. After the attack an increase occurs. In chronic gout careful study seems regularly to show a diminished excretion.

Diagnosis.—Typical attacks are easily recognized, but atypical cases

are often mistaken for rheumatic fever, chronic rheumatism or arthritis deformans (see Chronic Polyarthritis). (1) The patient's occupation and habits should be noted. In the United States barkeepers and brewery men are most often affected. (2) The location of the initial arthritis is often suggestive. (3) The presence of tophi in the ears, about the joints, or in the subcutaneous tissue is important. They must be distinguished from subcutaneous fibrous nodules. (4) The attacks of arthritis in gout may be afebrile. (5) Careful study of the urine may show a persistent low excretion of uric acid in chronic gout, or temporary diminution before an acute attack. No opinion can be based on single examinations, and, in fact, the uncertainties still existing with relation to the excretion of uric acid in gout render urinary examinations of very little service in diagnosis.

The BLOOD regularly shows a moderate leukocytosis (15,000–20,000) in an acute attack.

Treatment.—Prophylactic. Abstinence from alcohol, a careful regulation of the diet so as to avoid an excessive intake of nitrogen, moderate exercise in the open air, the free drinking of water, and regular hours should be advised for all children of gouty families.

Of the Acute Attack.—The patient is put to bed and given a milk diet. The bowels are opened by a laxative. Colchicum in doses of 20 minims of the wine or tincture of the seeds is given every two or four hours, for the relief of pain. If vomiting or purging is caused, it must be stopped. Aspirin, 10 to 20 grains every two hours, may be tried instead. The affected joint is elevated and wrapped in wool. Lead and opium wash or other anodyne applications may be used. After the first day or two a " purin-free " diet, of eggs, milk. butter, white bread, rice, sago, and cheese, may be allowed instead of milk alone.

Of Chronic Gout.—GENERAL.—A carefully regulated life, with diet and drink, exercise, bathing, hours of rest defined, is necessary.

DIET.—Many traditional restrictions and fads are still in vogue, but are being swept away by advances in accurate knowledge. Thus red meats are no more harmful than any other form of meat. Fruits are not objectionable. Vegetable or fruit acids are converted into alkalies in the body and are probably beneficial. Mineral waters are no more beneficial than any other, some of them may well be harmful. The diet must be restricted to the needs of the individual. It is to be general, meat being allowed in moderation. Only those foods known to be excessively rich in purin bases, such as meat extracts, sweetbreads, liver, kidney, or brain, fish roe, caviar, should be prohibited. Among the vegetables, cucumbers and tomatoes, and of the fruits, strawberries and bananas, are best avoided. The experience of an individual is often of more value than the accepted theories. If trial shows that any one article of diet brings on joint pains or distress it should be given up; if it can be taken without harm, it should be allowed.

DRINK.—Coffee, tea, and cocoa have long been forbidden because believed to contain purin bases in excess, but it is not proven that

caffeine or theobromine can be converted into uric acid and they may be allowed in moderation. Water should be drunk freely, particularly on rising and before meals. Any pure water can be recommended. No advantage has been proven to belong to any of the many mineral waters extolled as cures for gout. The mineral content of the water, whether of lithia or other salts, is of little moment. Alkalies as such are not of special value and alkaline mineral waters are not necessary. Exercise should be required, but never carried to the point of exhaustion. Walking, riding, and golf are especially suitable.

HYDROTHERAPY.—Not only the free drinking of water practiced at many springs, but the therapeutic application of water in baths, sprays, or douches is often very helpful. In the United States the Saratoga, Bedford, and White Sulphur Springs are recommended.

DIABETES MELLITUS

Definition.—A nutritional disorder characterized by a continued excess of sugar in the blood and its excretion in the urine, and by thirst and polyuria. The excreted sugar is grape-sugar.

Etiology.—The disease is relatively infrequent. In the United States in 1900 9.3 deaths per 100,000 of population were assigned to this cause. (1) Heredity plays a small part in its production, certain families showing a predisposition to the disease. (2) It occurs at all ages, but children are rarely affected, and its greatest incidence is in the decade from 50 to 60 years. (3) Men are affected more often than women, in the proportion of 4 to 3. (4) Fright, nervous excitement, or over-strain precede the onset in some cases. For this reason men of affairs, bank presidents and the like are often subject to this disorder. (5) Obesity is a frequent accompaniment, but Von Noorden regards it rather as a symptom of the underlying defect in metabolism than as a cause. (6) It may follow some of the acute infectious diseases, such as typhoid or scarlet fever, diphtheria, or rheumatic fever. (7) Organic lesions of the brain, such as tumors, general paresis, cerebral hemorrhage, may be followed by diabetes.

Morbid Anatomy.—The body of a diabetic patient is usually markedly emaciated, the skin coarse and dry. Boils or subcutaneous abscesses may be present. Not infrequently gangrene of a toe or foot is seen. The lungs are frequently the seat of tuberculosis, usually as a tuberculous bronchopneumonia. Acute lobar or bronchopneumonia may be found. The heart is hypertrophied and arteriosclerosis is marked. The liver may be cirrhotic and in rare cases cirrhosis of the liver is associated with pigmentation of the skin, the condition described by the French as cirrhose pigmentaire diabétique, by others as hemochromatosis. The pancreas shows lesions of some kind in about 50 per cent. of the cases. Chronic interstitial inflammation with atrophy and hardening of the gland is most common. Cysts, tumors, or calculi are found in other cases. Microscopically the pancreas shows interstitial inflamma-

tion with hyaline degeneration and atrophy, or complete disappearance of the islands of Langerhans. The latter is regarded as the essential lesion by Opie and other investigators. The kidneys are enlarged and show degeneration. Chronic nephritis may occur. The brain and nervous system frequently show lesions of varying character. Tumors are most frequent; cerebral hemorrhage, and degenerations of chronic type are also met with. The blood of the diabetic regularly shows a hyperglycemia.

Metabolism in Diabetes.—The disturbances of metabolism in diabetes affect mainly the carbohydrates. Of these three classes are distinguished: 1. Monosaccharides or glycoses ($C_6H_{12}O_6$) including grape-sugar (glucose) or dextrose, fruit-sugar (levulose), galactose and mannose. 2. Disaccharides or saccharoses ($C_{12}H_{22}O_{11}$) including cane-sugar (saccharose), milk-sugar (lactose), and maltose. 3. Polysaccharides or amyloses ($C_6H_{10}O_5$) including starch, glycogen, and dextrin. The carbohydrates, starch, cane-sugar, etc., taken in as food undergo a process of digestion and under the influence of the saliva, pancreatic juice, and succus entericus are converted into dextrose, which passing to the liver is converted by the liver cells into glycogen and there stored as such. Glycogen is in turn reconverted into glucose under the action of a liver ferment and so supplied to the blood. Glycogen and glucose are not derived from carbohydrates alone. A small amount of both can be formed from ingested proteid or the proteids of the body. Normally the blood contains from .1 to .2 per cent. of glucose. In diabetes there is always an excess, a hyperglycemia, amounting in some cases to as much as .6 or even .7 per cent. One cause of this hyperglycemia is that the liver has lost to some extent its power of storing up as glycogen the dextrose supplied to it by the food or the proteids of the body. A second element in the production of the hyperglycemia is that certain tissues of the body, especially the muscles, have lost, to some extent at least, their power of oxidizing the glucose of the blood and converting it into water and carbon dioxide. Evidence has been supplied that this loss of oxidizing power in the muscles is not due to defect in the muscles themselves, but to the absence of a certain glycolytic substance (possibly a ferment) normally supplied by the pancreas.

The glycogenic function of the liver is undoubtedly subject to the control of the central nervous system, as is shown by the glycosuria which follows experimental puncture of the floor of the fourth ventricle in dogs. This nervous control of the glycogenic function of the liver would explain the relationship between nervous diseases and diabetes. On the other hand, the function of the pancreas in supplying a ferment or glycolytic substance which aids the muscles in the assimilation of glucose explains the frequency with which pancreatic lesions are found to be associated with diabetes, and the glycosuria resulting from the experimental removal of the pancreas.

It would thus appear that diabetes may result from a variety of conditions and that no one lesion will explain all cases of the disease.

Symptoms.—The invasion of the disease is insidious. (1) **Loss of weight** associated with languor and weakness usually first attracts attention. Some patients, especially the obese, may not lose weight at all, but as a rule the loss is steady and progressive. (2) **Thirst is more** active than usual and may be insatiable. (3) The appetite is similarly exaggerated and may be enormous. Some patients suffer from gastric distress, eructations, and constipation, but in most cases the digestion

Fig. 59.—*A*, diabetic gangrene of the great toe; *B*, diabetic gangrene of the great toe.

is excellent. (4) Polyuria is marked. In moderate cases three or four quarts may be voided, while the output amounts to as much as 5 to 10 quarts in the severer forms of the disease. (5) The skin becomes dry and harsh, the mouth parched, the tongue red and glazed. (6) The presence of sugar in the urine is the essential symptom. The urine is pale, clear, of specific gravity varying from 1024 to 1045, and usually free from albumin or casts. With nephritis albumin and casts appear.

Sugar is present in varying amounts, averaging 2 or 3 per cent. In severe cases it may amount to 8 or even 10 per cent. The total output of sugar in 24 hours varies from a few grains to 1000 or even 1500 grains. Sugar is demonstrated by Fehling's test and estimated quantitatively by the same test, by fermentation, or the polariscope. The fermentation test should always be used to confirm the presence of sugar in the urine, for there are a number of other substances which reduce Fehling's solution, such as glycuronic acid, homogentisic acid, uric acid in excess, lactose, and kreatinin. Acetone, diacetic acid, and β-oxybutyric acid may also be found in the urine, especially in severe cases, by appropriate tests.

These are the common symptoms of the disease; various others may be due to involvement of special organs or to complications of the disease. (1) THE SKIN.—Boils or carbuncles are common and in the impaired state of the diabetic may become grave complications. Eczema which may be very annoying may be produced on the genitals, especially of women, by the irritation of the urine. Gangrene of the foot or other part may occur (see Fig. 59). (2) RESPIRATORY SYSTEM.—The breath often has a sweetish odor, resembling that of acetone. Acute tuberculosis, lobar or bronchopneumonia or gangrene of the lung may develop. (3) CIRCULATORY SYSTEM.—The arteriosclerosis may be the cause of symptoms. It underlies the gangrene of the extremities so often seen, and may give rise to symptoms of nephritis or myocarditis, or cause cerebral hemorrhage. (4) NERVOUS SYSTEM.—Headaches may be a marked symptom. Neuralgia or neuritis frequently adds to the patient's distress. Sexual power is usually lost. Perforating ulcer of the foot is not rare. Degeneration of the posterior columns of the cord presenting symptoms resembling tabes occurs at times. (5) CATARACT is not uncommon in the elderly patients. Retinitis, atrophy of the optic nerve, or paralysis of accommodation may develop. (6) DIABETIC COMA is the most serious complication, being regularly fatal. The type of coma varies. (a) Coma with dyspnea, Kussmaul's air-hunger type, is most frequent. (b) An alcoholic type with symptoms resembling intoxication. (c) Coma with collapse, in which the pulse becomes rapid and feeble, the surface livid, the extremities cold. The approach of coma may be indicated by lassitude, headache, or restlessness and excitement. Drowsiness develops and deepens into coma, from which, as a rule, the patient never rouses. Diabetic coma has been proven to be the result of the acid intoxication caused by the β-oxybutyric acid, and its presence in any considerable amount is always a warning of the approach of coma. When β-oxybutyric acid is present, acetone and diacetic acid are also found, and because of the difficulty surrounding the tests for β-oxybutyric acid, acetone and diacetic acid are first tested for. If present, β-oxybutyric acid must be tested for, but if there is no acetone or diacetic acid, we may conclude that β-oxybutyric is not present.

Course and Prognosis.—In children diabetes is usually swiftly fatal. Adults also sometimes succumb quickly, but as a rule the course

of the disease is protracted over several years. The older the patient the less effect diabetes seems to have. Obese people also seem to bear the disease well.

Diagnosis.—This depends chiefly on the examination of the urine. The sugar must be shown to be persistently present, and proven to be grape-sugar, to justify the diagnosis. Temporary glycosuria from any of the following causes must be excluded: (a) narcosis from ether, alcohol, or opium, or coma from any cause; (b) carbon dioxide, amyl nitrite, mercury, or strychnine poisoning; (c) epilepsy, hysteria, or neurasthenia; (d) chlorosis or exophthalmic goitre; (e) acute infectious diseases. If the characteristic symptoms of diabetes be absent some days of observation may be required to determine the question. The possibility of deception by the addition of sugar to normal urine must be considered in some cases.

Treatment.—HYGIENIC.—A carefully regulated life, free from worry or care, is necessary. Daily lukewarm baths serve to prevent some of the skin complications. Moderate exercise, if the patient's condition permits it, is desirable. Constipation must be corrected. DIETETIC.— The hope of cure in diabetes lies in the proper management of the diet. The principle upon which the diet is managed is that if the patient can take even the smallest amount of carbohydrate food without excreting sugar, the tolerance of carbohydrates can be raised gradually, the amount of carbohydrates well-borne can be increased and ultimate cure may be brought about. In the beginning the amount of sugar excreted, while the patient takes an ordinary mixed diet, should be determined for several successive days. The urine should also be tested by fermentation to confirm the findings of other tests. The patient should then be put upon a diet free of starches and sugar, such as the following:

Breakfast: 7.30 A.M. 120 grams beefsteak or mutton chops, two boiled or poached eggs, 200 c.c. coffee or tea without sugar.

Lunch: 12.30 P.M. 200 grams cold roast beef, mutton, or chicken; 60 grams of celery, cucumbers or tomatoes with 5 c.c. vinegar, 10 c.c. of oil, pepper and salt as required; 20 c.c. whiskey; 400 c.c. water; 60 c.c. coffee.

Dinner: 6 P.M. 200 c.c. clear bouillon; 200 grams roast beef; 60 grams lettuce with 10 c.c. vinegar, 20 c.c. of olive oil or ahree table-spoonfuls of some green vegetable, such as spinach; 20 c.c. whiskey (if desired); 400 c.c. Apollinaris or water.

Supper: 9 P.M. 2 eggs raw or cooked; 400 c.c. Apollinaris or water.

About 15 grams of butter may be given daily upon the eggs or in gravies. Saccharin may be substituted for sugar. No milk or cream is to be allowed.

If the examination of 24-hour specimens of urine shows no sugar for several days, the disease is of mild type and starches may be added, 30 grams of bread being allowed the first day and the amount later increased if no sugar appears. Later potatoes and other vegetables may be added. If sugar continues to appear in the urine on the re-

stricted diet, Naunyn advises a fast-day. If the amount of sugar is small this will sometimes cause it to disappear altogether, and tolerance for minute amounts of carbohydrates may then be tried. If persistent trials fail to free the urine of sugar, carbohydrate food must be allowed in moderate amounts, as these patients do badly if restricted too long to the proteid diet, and the danger of coma is increased. In these cases trial must be made to find just what amount of carbohydrates must be allowed to maintain strength and nutrition and not too greatly increase the amount of sugar in the urine.

A great variety of medicines have been given in diabetes, but none has any definite effect upon the process. Opium and codeine have been especially used. They serve to lessen distress or pain, but they increase the tendency to constipation, and their administration should be avoided, unless required for the relief of suffering. Pancreatic extracts have been given, but without success. Arsenic in the form of Fowler's solution, or as arsenious acid, has been much praised. Sodium bromide, phenacetin, the salicylates and many other drugs have been employed at various times, but no confidence can be placed in their effects.

TREATMENT OF DIABETIC COMA.—The presence of β-oxybutyric or diacetic acids in the urine must always be regarded with concern, although occasionally a patient's urine shows their presence for many weeks without harmful results. In moderate cases, when these acids appear, effort should be made to reduce them by increasing the amount of carbohydrate food, especially if the intake of starches and sugars has been much restricted. A moderate increase in the allowance may cause the disappearance of the acids. Since it has been shown that the harmful acids are derived not from the carbohydrates, but the fats, the latter should be materially reduced or excluded from the diet. Subcutaneous injections of levulose in 5 to 10 per cent. solutions have recently been recommended by Von Noorden. Others give levulose by mouth. Sodium carbonate or bicarbonate must also be given by mouth, rectum or subcutaneously, in very large amounts. From 1 to 2 drams hourly by mouth, and twice as much by rectum may be given. For intravenous injection a 3 to 5 per cent. solution, carefully sterilized, is used. From 1 to 2.5 ounces of sodium bicarbonate have been given at one injection. Threatened coma may be temporarily relieved by these measures, but the fatal outcome cannot be long delayed.

Pruritus is best treated by reducing the amount of sugar in the urine. Lotions of boric acid or hyposulphite of soda (30 grains to the quart) may give temporary relief. Boils, carbuncles, or gangrene must be treated aseptically. Operations are likely to induce coma, but the danger of septic infection is great.

DIABETES INSIPIDUS

Definition.—A rare disorder characterized by the persistent passage of large quantities of urine free from sugar, albumin, or casts.

Etiology.—Young people are most often affected. 1. Various affec-

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tions (a) Trauma or tumors of the brain, especially those involving the floor of the fourth ventricle, may be the cause. (b) Cerebral syphilis has preceded some cases. (c) Hysteria, epilepsy, or neurasthenia may accompany the polyuria. 2. The disease may be idiopathic, no cause being ascertainable. The majority of cases belong in this group.

Morbid Anatomy.—No constant pathological changes are found. The brain lesions mentioned above may be present. The kidneys and bladder are sometimes enlarged.

Symptoms.—Polyuria is the essential symptom. Enormous quantities of urine are passed, amounting in some cases to 15 or 16 quarts. The urine is consequently water-like, its specific gravity 1001–1005, almost colorless. It contains no sugar, no albumin or casts, as a rule, although traces of albumin may at times appear. Increased thirst and appetite are commonly associated. Nutrition is usually unimpaired, but sometimes suffers severely. The temperature is subnormal. The COURSE of the disease is usually very chronic, the patients ultimately become exhausted and die in coma.

Diagnosis.—Diabetes mellitus is excluded by the examination of the urine; chronic interstitial nephritis by the urinary conditions and the absence of arteriosclerosis and hypertrophy of the heart. A distinction cannot always be drawn between a symptomatic polyuria and diabetes insipidus. In fact, some writers exclude the polyuria of hysteria or epilepsy from the disease, others include it. If the condition is persistent it may be termed diabetes insipidus.

Treatment.—Codeine or opium may be given to lessen thirst and thus reduce the polyuria. Valerian is more often prescribed, in 5 grain doses of the powder or 15 grains of the valerianate of zinc three times a day. The doses may be gradually increased. If cerebral syphilis is suspected specific treatment is in order.

RACHITIS
(Rickets)

Definition.—A disease of infancy and childhood caused by defective nutrition, with its most striking manifestations in the growing bones.

Etiology.—The disease regularly develops between the ages of 3 months and 3 years. Both sexes are affected. While it is much more common among the children of the poor in large cities, it is frequently met with in country children and in those of wealthy parents. Whatever influences lower the vitality of children, such as illness of the mother during pregnancy or lactation, poverty, overcrowding, lack of sunlight, acute disease, especially of the digestive organs, predispose to the development of rickets. The essential cause, however, is defective nutrition. This may be brought about either by improper feeding or by defective assimilation of proper food because of inherent weakness of digestion and assimilation. (a) Improper feeding is by all odds the most common cause. Nursing children very rarely develop rickets. Artificially-fed children frequently do so. Artificial foods containing

an excess of carbohydrate materials, condensed milk, or cow's milk too greatly diluted, are the foods upon which rickets is most often developed. (b) Defective digestion or defective assimilation is apparently the cause of rickets in some children who have been most carefully fed. Such defective digestion is most often due either to inherent feebleness of the child or to intercurrent diseases of the alimentary tract, such as ileocolitis.

Morbid Anatomy.—The most striking changes are in the bony skeleton. The head is large, the frontal bosses prominent and the skull at these points thick. Craniotabes is often present in the occipital regions where the bones are subjected to pressure. The maxillæ are usually small and imperfectly developed. The teeth are late in appearing. The thorax often shows lateral grooves along the line of attachment of the diaphragm with flaring of the free borders of the ribs below. The junctions of the ribs and cartilages are regularly enlarged, forming the so-called rachitic rosary. The abdomen is prominent; the muscles, both of trunk and extremities, flabby. The long bones are frequently deformed, the lower extremities showing bow-legs or knock-knee, the upper, abnormal curvature of the humerus, radius and ulna. The clavicle is often unduly curved. The spine is markedly curved and may appear kyphotic. The junctions of epiphysis and diaphysis of the long bones, especially at the wrist, ankle, and knee, are regularly enlarged. Bronchitis or bronchopneumonia is almost invariably present post-mortem. The liver and spleen are sometimes enlarged. The characteristic lesion of rickets is found in the costo-chondral junctions or the junction of epiphysis and diaphysis of the long bones. In rickets this junction is enlarged both laterally and longitudinally, and instead of the normal sharp line of demarcation between the two the line is wavy and indistinct. These changes are visible to the naked eye. Microscopically the zone between epiphysis and diaphysis is found unduly vascular and with a very irregular distribution of cartilage cells and islets of calcification. The cancellous tissue of the shaft is also unduly vascular and ossification is imperfect, so that the bones are soft and easily deformed. The subperiosteal bone formation shows a similar vascularity and imperfect ossification, so that some areas (frontal bosses) are thickened, and others unduly thin (occipital bones).

Symptoms.—GENERAL.—While the skeletal signs of rickets are always the more impressive symptoms, the constitutional effects of the disease are the more important. Head-sweating and restless tossing of the head are common, so that the hair over the occiput is worn thin by friction. Mouth-breathing due to adenoids is common, and the lymphatic nodes are generally enlarged. The rachitic child suffers from frequent attacks of bronchitis or bronchopneumonia. The digestion is impaired, with flatulence and constipation usually present. In some cases ileocolitis with diarrhea develops. The general nutrition suffers little and rachitic children often appear well-nourished or fat, but the tissues, especially the muscles, are flabby. The children are consequently late

in walking. The teeth are late in appearing. Resistance to infection is lowered and rachitic children succumb easily to acute infectious diseases, especially to measles or diphtheria.

SKELETAL.—These have been described under the morbid anatomy. The open fontanel, prominent frontal bosses, small jaw-bones, the curvatures of the long bones, the rachitic rosary are all seen in marked cases. In the milder cases only the beading of the ribs, the delayed dentition, or open fontanel indicates the presence of the disease. Pelvic deformities not of importance during childhood, but of gravest moment in adult women, are common. Owing to contraction of the pelvis, enlargement of liver and spleen and distention of the intestines by gas, the abdomen is regularly prominent and tympanitic.
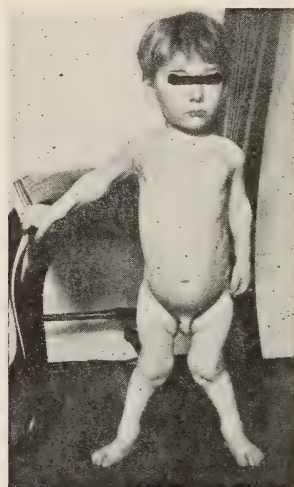
FIG. 60.—Achondroplasia. Note the large head, the comparatively normal body, the shortness of the extremities, particularly the upper arms and thighs, and the deformities. From the collection of Dr. W. P. Northrup.

Course and Prognosis.—Rickets, rarely fatal in itself, leads to high mortality through death from complicating bronchitis or bronchopneumonia or acute infectious disease. Those who survive the disease recover slowly with more or less permanent deformity of the skeleton. The great majority of cases of knock-knee and bow-legs are due to rickets.

Diagnosis.—The rachitic rosary, delayed dentition, open fontanel, and enlarged epiphyses, without the advanced skeletal changes, make the diagnosis easy if these signs are looked for.

Treatment.—HYGIENIC.—Fresh air and sunlight are of great value. Systematic bathing, a bath of 85°, followed by sponging with slightly cooler water, should be practiced. Massage may be given. The child must be kept off the feet until the bones have become strong enough to bear the weight.

Dietetic.—Change of food is the essential measure. Carbohydrates, if in excess, must be reduced and fat given. Breast milk is ideal for the young. In older children a proper dilution of cow's milk in which the percentage of fat is steadily increased is given. Cod-liver oil may also be prescribed, most easily in a malt extract. Phosphorus is highly commended, 6 to 15 drops of the elixir (U. S. P.) three times a day.

ACHONDROPLASIA

Definition.—A developmental disease which impairs the cartilaginous ossification, especially in the long bones.

Etiology.—Heredity plays an important rôle. The disease has repeatedly been observed in successive generations. Several members of one family may likewise be affected, especially in the case of very large families.

Of the nature of the disease little is known. It presents certain resemblances to rickets, syphilis, and cretinism, which have been regarded as indicating relationship by various writers, but our knowledge of the disease at present leads to its classification as a separate clinical entity, but does not enable us to exactly determine these questions.

Morbid Anatomy.—The lesions are found in the bones, especially the long bones. The extremities are short, the shortening affecting chiefly the proximal segments, arm and thigh. The trunk is practically normal, although there may be a moderate enlargement of the costochondral functions reproducing the rachitic rosary. The head is large with prominent bosses. Premature synostosis of the bones surrounding the foramen magnum and likewise of the junction of the occipital and sphenoid bones is found. The vertebræ are normal.

Microscopic examination shows the fundamental error to be defective cartilage formation. Thus the rosary is not due, as in rickets, to swelling of the cartilage, but to defective development of cartilage with excessive formation of bone in the rib-end, so that the cartilage may lie in a bony cap.

The cartilage is abnormal throughout, thinner than it should be, more fibrous, sometimes vacuolated, with an absence of the normal zone of proliferation, hence the name achondroplasia.

In some instances the epiphyses are almost entirely absent, and the shortness of the long bones is readily explained.

Symptoms.—Most achondroplasiacs are still-born and the disease is best known from the studies of such skeletons. Those that survive constitute one of the well-recognized types of dwarfs. The essential features are the large head with prominent bosses, the almost normal trunk, with the disproportionately short extremities, the shortening affecting especially the arm (humerus) and thigh (femur). Deformities of the extremities, especially knock-knee, are common (see Fig. 60). The hands are small, short, and cubical, the fingers being all of nearly the same length. The fingers often diverge like the prongs of a trident.

Teething, talking, and walking are not delayed as in rickets, and the mentality of most of these dwarfs is normal. Some of them are unusually bright.

Diagnosis.—This must be based in the living upon the skeletal characters above noted. The affection must be distinguished from the others forms of dwarfism, especially infantilism, cretinism, and rickets.

Infantilism is marked by the failure of growth with preservation of the normal relations in length and size of parts, and absence of the secondary sexual characteristics. Achondroplasiacs have the skeletal abnormalities, but develop sexually.

Rickets is distinguished by the abnormally large epiphyses, the deformed extremities, which are, however, of normal relations as to length, the wide-open fontanel, the delay in teething, walking and talking.

Cretins are easily distinguished by the characteristics noted on page 272.

Treatment.—No treatment is known to have any influence upon the disease, but careful feeding, massage, passive movements, and electricity are indicated.

OBESITY

Definition.—A metabolic disorder characterized by the deposition of excessive amounts of fat.

Etiology.—(1) Heredity plays an important rôle. The tendency to obesity is transmitted in certain families, especially in those subject to gout. (2) The disorder sometimes dates from birth, but usually appears between the ages of 30 and 50 years. (3) Women are more often affected than men. In women repeated pregnancies seem to favor obesity and the increase in weight frequently dates from child-birth or miscarriage. (4) Persons who lead a quiet, sedentary life are most subject to the disorder. (5) The prime factor is the habitual ingestion of excessive amounts of food, especially carbohydrates and fats, and often also the use of malt liquors, or sweet wines.

Metabolism in Obesity.—The essential feature of obesity is the ingestion of more food than can be oxidized and turned into energy. Carbohydrates and fat most readily yield fat, but fat can also be formed from proteid foods. The problem of obesity is for the most part a purely mathematical one between the energy required by the body and that supplied by the food. A peculiar tendency to the formation of fat, based upon disordered metabolism, is claimed to exist in certain individuals, so that they increase their fat while living very abstemiously. While this has never been thoroughly proven, it is generally accepted that such a disorder of metabolism, essentially a diminished power of oxidation, does exist. Anemia lessens the oxidation power and is not an infrequent accompaniment of obesity.

Morbid Anatomy.—Fat is found especially in the subcutaneous tissues, particularly those of the abdomen, in the omentum and mesen-

tery, about the heart and kidneys, and in the liver. The heart is enlarged, the right side dilated, and much fat is found on the surface and also infiltrating the heart muscle. The liver is very fatty.

Symptoms.—Obesity is consistent in some cases with perfect health and great activity. In most instances the increasing weight leads to diminished activity, because every effort demands greater exertion, and the subjects become inactive both mentally and physically, and this inactivity still further increases the tendency to obesity. Obesity is regularly accompanied by diminished power of resistance to disease or shock, and very fat persons are poor subjects for acute infections, such as typhoid fever or pneumonia, or surgical operations. In some cases the fatty infiltration of the heart produces symptoms of feebleness and irregularity of the heart action (see Fatty Heart).

Treatment.—Alcohol must be forbidden and the total amount of food and drink reduced. Banting reduced the total diet to from 21 to 27 ounces, of which 13 to 16 were of animal food, and only 2 of bread. Sugar and other starches were excluded. Ebstein would allow fats because they produce satiety. Ebstein's dietary is as follows:

Breakfast: Toasted white bread, 2 ounces. Tea without milk or sugar, 8 to 9 ounces.

Dinner: Soup made with beef marrow. Fat meat, 4 to 5 ounces, and a moderate allowance of spinach, beans, peas, cabbage, or asparagus. Light white wine, 2 or 3 glasses. Tea, as before.

Supper: An egg, a little roast meat, with fat. Bread, 1 ounce, with butter. Tea, as before.

Oertel's diet would allow less fat and more proteid, and is ordered as follows:

Morning: Coffee or tea with milk, 6 ounces. Bread, 3 ounces.

Noon: Soup, 4 ounces. Roast beef or other meat, 7 to 8 ounces; salad or light vegetable; a little fish; bread or other farinaceous pudding, 1 ounce; fruit, 3 to 6 ounces.

No liquids except in hot weather, 6 ounces of light wine.

Afternoon: Coffee or tea, 6 ounces. Occasionally bread, 1 ounce.

Evening: Eggs, 1 or 2; bread, 1 ounce; perhaps a small bit of cheese, a little salad, and fruit; light wine, 6 to 8 ounces, with 4 or 5 ounces of water. The amount of water is restricted to 1 pint.

Such a dietary is regularly combined with progressive exercise, the patient being required to walk carefully graduated distances, involving increasing effort, either on the level or on hills. Hot baths, even the Turkish bath, may be employed in certain cases.

VII

INTOXICATIONS AND MISCELLANEOUS DISEASES

ALCOHOLISM

INEBRIETY OR DRUNKENNESS

Definition.—An acute or chronic intoxication due to the excessive use of alcoholic drinks.

Etiology.—(1) Heredity plays an important rôle, probably through the transmission of an inherently weak and unstable nervous system. (2) This inherent weakness of the nervous system is the basis of excessive indulgence in most cases, and for this reason alcoholism, both acute and chronic, is now looked upon as a disease rather than a habit. (3) Many other factors enter into the causation of alcoholism in individual cases, such as the force of example, social custom, the fatigue of exhausting labor, a false belief that the use of alcohol gives strength, and the like. Occupations involving the handling of liquors are especially perilous.

Symptoms.—(1) ACUTE ALCOHOLISM.—The ingestion of alcohol causes a primary stimulation, soon followed by marked fatigue and exhaustion, affecting chiefly the circulatory and nervous systems. The face flushes and the pulse becomes rapid and bounding. Stimulation of the cerebral cortex is shown by unusual animation and the rapid flow of ideas. Peculiarities of disposition are accentuated and the individual becomes more affectionate or quarrelsome. Muscular incoordination soon develops, the victim staggers and loses control of his movements. The mind becomes dulled, stupor, and finally coma supervene. In this state the face is flushed, the pupils dilated, normal, or contracted, but not unequal; the pulse rapid, full and bounding; and the breath smells strongly of alcohol. Muscular twitchings are not uncommon, and general convulsions may occur in certain persons. The alcoholic coma is rarely profound; the patient can be roused by slapping the face, or steady pressure over the supra-orbital nerves, and will then move all his extremities, struggling and using abusive language. In alcoholic coma the temperature is often subnormal, sometimes below 95°.

Diagnosis.—Difficulty in the recognition of acute alcoholism is rare. Two sources of error should be avoided: (1) Persons suffering from other severe affections may be given sufficient alcohol to taint the breath. (2) Drunken persons by falling or being struck frequently suffer severe injuries, such as fracture of the skull, the symptoms of which may be concealed by the intoxication. (For the differential diagnosis of alcoholic coma see Uremia.) Grave errors are frequently made by ambulance or police surgeons in large cities. To avoid these the only safeguard lies in having every comatose patient cared for and observed carefully until the diagnosis is established beyond doubt, or he has fully roused from the coma.

Treatment.—The stomach should be thoroughly emptied, by washing if feasible. The patient may then be allowed to sleep off the stupor. A dose or two of aromatic spirits of ammonia, one-half to one dram, often aids in recovery. In severe cases with subnormal temperature external heat and cardiac stimulants (caffeine or strychnine) may be necessary.

(2) CHRONIC ALCOHOLISM.—Constant or periodic excess in the end leads to the condition denominated chronic alcoholism. The habitual use of alcohol is a common cause of chronic gastritis, cirrhosis of the liver, chronic nephritis, arteriosclerosis, and polyneuritis, and many cases of chronic alcoholism present one or more of these conditions. The more characteristic symptoms are referable to the influence of alcohol upon the nervous system. In the brain, cord, and peripheral nerves chronic inflammatory and degenerative changes are demonstrable. The distribution of the lesions and the symptoms resulting therefrom vary in different cases and several clinical affections have been described.

Symptoms.—Many persons use alcoholic beverages in excess for years without serious symptoms. Others show tremors of the hand and tongue, increasing mental dulness and apathy, forgetfulness, irritability of temper, disregard of duty and all moral obligations, and finally complete moral and physical degeneration. The facies of the chronic alcoholic patient is often characteristic. The eyes are watery, the conjunctivæ congested, the nose and cheeks deep red with prominent dilated veins, the expression dull and the speech slow and imperfect. Various forms of insanity, especially dementia paralytica, may end the course.

Treatment.—Control, moral and possibly physical, is of prime importance. Institutional treatment is therefore often necessary. The organic lesions of the patient, gastritis, nephritis, etc., must be appropriately treated, and every effort made to restore bodily vigor by diet and exercise. For the habit itself the hypodermic administration of atropine and strychnine, gr. 1/100 each, three times a day, with gradual reduction of the dose, has been found useful. The Towns-Lambert method of treating drug habits may be employed.

In treating an alcoholic the purgatives (see p. 300) and the initial dose of the belladonna mixture are given at the same time. The belladonna mixture is continued day and night. At the 12th, 24th, and 36th hours the cathartics are repeated. After the appearance of characteristic bilious green stools, the castor oil is given, about the 44th or 45th hour.

Occasionally the treatment is continued over an additional period, the cathartics being given at the 48th hour and the treatment continued till 60 hours have elapsed.

Alcohol is given only to elderly or very nervous patients, who are allowed four or five doses of whiskey, 2 ounces each, during the first 24 hours. Sedatives are often required to produce sleep and cardiac stimulants are given after the first 24 hours, or earlier, if necessary.

DELIRIUM TREMENS.—This affection occurs in the course of chronic alcoholism (1) following a debauch or (2) after an injury, a surgical operation, or acute illness. Delirium tremens is frequently seen after severe fractures, or operations of any kind, or complicating pneumonia, erysipelas, and like acute infections.

Symptoms.—The attack begins with restlessness, insomnia, and headache. The tremor of fingers and tongue is marked. The tongue is coated and the appetite lost. Soon the characteristic hallucinations develop, the patient seeing snakes, rats, mice, and hearing angels or demons speaking to him. An active delirium follows in which the patient struggles to escape from bed, talking or shouting incessantly, grasping or striking at imaginary creatures in the air or upon the bed, his eyes expressing wild excitement or fear. Very rarely the delirium is of pleasant character. The temperature is raised, the pulse rapid and bounding, or later weak and small. The delirium after three or four days subsides gradually or the patient succumbs to exhaustion.

Diagnosis.—The tremor of hands and tongue, and the hallucinations are characteristic. Careful physical examination is essential to exclude the presence of injury, such as fracture of the ribs or an extremity, pneumonia, erysipelas, or other intercurrent disease.

Treatment.—Restraint is necessary and is best effected by careful attendants, otherwise by sheets or straps which confine the arms and legs, while not restraining the movements of the body or respiration. Rest and food are the essentials. Chloral and bromide of soda are given in doses of ten to fifteen grains of the former to twenty or thirty of the latter every two or three hours. Paraldehyde in doses of one to two drams by mouth or rectum is valuable. Hyoscine hydrobromate, gr. 1/100, may be given occasionally. Morphine is rarely satisfactory and always dangerous in this condition, too great respiratory depression being produced by doses sufficient to quiet the patient. Milk, broths and eggs should be given as freely as possible. A continued hot bath is advised for the delirium, where circumstances permit sufficient attendants to carry out the procedure.

SEROUS MENINGITIS OR WET-BRAIN.—Following a debauch or an attack of delirium tremens alcoholic patients sometimes develop the so-called alcoholic or serous meningitis. More accurately speaking, it is an edema of the pia mater. The patient sinks into a stupor, with stiff neck, somewhat contracted pupils, hyperesthesia of the skin, and sensitiveness to pressure of the extremities, and sometimes rigidity, the temperature normal or slightly raised, the pulse rapid and weak. After some days the patient improves or the stupor deepens and he succumbs to exhaustion or intercurrent pneumonia. The condition may last three or four weeks.

Treatment.—Careful feeding is important. Strychnine, gr. 1/60–1/30, and ergot, 30 minims of the fluidextract or its equivalent, should be given hypodermatically every two hours. Camphor and caffeine may also be used.

OPIUM POISONING AND MORPHINISM

ACUTE POISONING.—An overdose of opium or morphine produces a very characteristic picture of increasing drowsiness ending in coma, with pupils contracted to the last degree (pin-point), respiration falling to 12, 10, or even 6 a minute, and finally ceasing with death from asphyxia.

Treatment.—The stomach should be washed out with a 1/500 solution of potassium permanganate, since morphine even when taken hypodermatically is excreted in part by the stomach, and the permanganate has been shown to prevent the toxic action of opium or morphine. The washing is repeated if the poisoning is severe. Atropine, gr. 1/100 or more, is given as an antidote, and repeated as indicated. Black coffee is given by rectum. Artificial respiration must be kept up for hours, if necessary. For this purpose the Fell-O'Dwyer apparatus is invaluable.

MORPHINE OR OPIUM HABIT

Definition.—A chronic intoxication with opium or one of its derivatives, usually morphine.

Etiology.—(1) A fundamental weakness of the nervous system, as in other evil habits, is present in many cases. (2) The influence of temptation is seen in the fact that druggists, physicians and nurses are so frequently victims of the habit. (3) Painful affections, such as neuralgias, dysmenorrhea, endometritis, etc., or insomnia are often the explanation of the beginning of the habit. The use of morphine or opium for the relief of any distress likely to be persistent is therefore attended with great danger.

Symptoms.—Opium or morphine is occasionally used for some time without serious symptoms. Most persons give early evidences of the habit. These are first seen in the alternations of the exhilaration produced by the taking of the drug and the depression which naturally follows. Immediately following the indulgence the habitué is stimulated, the eyes are bright, the pupils contracted, the cheeks flushed, the mind alert and active, and the appetite increased. Itching of the nose requiring frequent rubbing is often notable. As this effect wears off the individual becomes dull and drowsy, the pupils dilate, the patient is morose and irritable, without energy or inclination to exertion of any kind. These alternations present themselves more and more frequently, requiring larger doses and more frequent administrations to satisfy the longing for the drug. Gradually a condition of malnutrition develops, the patient loses appetite altogether, suffers from chronic constipation, the hair becomes gray, the skin dry and parchment-like, and flesh and strength fail. If the drug is taken hypodermatically, abscesses frequently develop in the various punctures. But the most striking effect of the habitual use of opium is, in most cases, the total failure of the

moral nature. The victims become most adept liars, and unable to tell the truth. They become oblivious to all ordinary moral impulses, but are rarely, like the alcoholic, abusive or destructive.

Diagnosis.—The alternation in the condition of the patient from time to time is suggestive of a drug habit. The contracted pupils and itching of the nose point to opium or morphine. The marks of the hypodermic needle can often be found.

Treatment.—Because of the deception practiced by patients and their absolute untrustworthiness and inability to tell the truth, the treatment of these patients outside of institutions where they can be kept under constant supervision and controlled to some extent is regularly futile. The individual who really desires to escape from his habit and will honestly co-operate with his physicians is rare indeed. The principles of the treatment in institutions are the gradual withdrawal of the drug, the relief of distress and craving by ergot and bromides, hot baths or cold packs, and the building up of the patient by careful feeding and tonics, especially nux vomica or strychnine.

The Towns-Lambert method of treatment of drug habits has been widely tested and found effective. The essentials of the treatment of morphinism consist in the thorough purgation of the patient, followed by the administration of fractional parts of the customary daily dose of morphine at stated intervals and also of a mixture composed of 15 per cent. tincture of belladonna, two parts, and one part each of the fluidextracts of hyoscyamus and xanthoxylum. The treatment is outlined as follows: At the outset the patient is purged by the administration of five compound cathartic pills and five grains of blue mass, followed, if necessary, by a saline after six or eight hours. After three or four movements two-thirds of the daily allowance of morphine is given in three divided doses (2/9 each) at one-half-hour intervals. Eighteen hours later one-half of this dose is administered, and again after eighteen hours one-half of this dose (one-sixth the original dose). Ten hours after each dose of morphine the purgation is repeated as at the outset, and finally about the fifty-sixth hour of treatment two ounces of castor oil are given. The belladonna mixture is given day and night, beginning with doses of six drops hourly, and increasing the dose two drops every six hours till sixteen drops are given, the mixture being diminished or stopped if symptoms of belladonna poisoning appear.

Among the symptoms of poisoning noted are dilation of the pupils, a dryness of the throat or redness of the skin, a peculiar incisive or insistent voice, and insistence on one or two ideas. The mixture is renewed at a lower dosage on the subsidence of these symptoms, but is continued throughout the treatment. Nervousness and discomfort are treated by hypodermic injections of codeine, in doses sometimes as large as five grains, repeated as necessary. Stimulants, such as strychnine and digitalis, are given about the thirtieth hour of treatment, and after the withdrawal of the drug tonics, such as phosphorus and arsenic. Careful feeding is necessary in the after-treatment.

COCAINE POISONING

Acute poisoning is rarely seen except from excessive doses in surgery. Nervous excitement, rapidity and weakness of the pulse, with pallor and finally collapse, may be produced. The treatment is purely symptomatic, by sedatives and stimulants as required.

COCAINISM OR COCAINE HABIT

The habitual use of cocaine is increasing. It is rarely taken alone, but the subjects often indulge in morphine, alcohol, or other drugs.

Etiology.—(1) The habit is frequently developed from the use of nostrums advertised as catarrh cures and containing cocaine in quantity. (2) The drug may be taken for the relief of physical exhaustion or pain. (3) In the large cities the taking of cocaine has become one of the established modes of debauchery.

Symptoms.—These are difficult to detect or describe because the cocaine is so often used together with other drugs. Alternating periods of exhilaration with increased mental and physical activity and depression with irritability and sluggishness occur, and become more and more frequent. The exhilaration usually follows an opportunity to take the drug and by being alone. The victims develop the unreliability of character seen in other drug habits, become slothful, indolent, anemic and poorly nourished. Not infrequently the habit results in insanity, with hallucinations and delusions.

Diagnosis.—This is difficult, unless the drug is found in the possession of the patient or acknowledged by him. The alternations of exhilaration and depression and the changes in character are suggestive of drug-taking. The cocaine-taker seeks to be alone and is very secretive regarding his habits, in this differing from the alcoholic; on the other hand, the contraction of the pupils and the itching of the nose characteristic of morphine are not observed.

Treatment.—This, as in morphine taking, is best done in institutions, and for the same reason. The drug is withdrawn gradually and sedatives or stimulants administered as required, while every effort is made by careful feeding and nursing and hydrotherapy to improve the general condition.

Cocainism may be treated in the same manner as morphinism (see p. 300), except that no cocaine is given and cardiac stimulants are administered from the beginning.

CHRONIC LEAD POISONING

(Plumbism. Saturnism)

Etiology.—The disease is produced through the taking in of lead either by the alimentary or respiratory tracts or by the skin. There is no special susceptibility of age, race, or sex, but men are most often

affected by reason of exposure. Any calling involving the handling of lead ores, or any of the common lead products is dangerous. Miners, painters, plumbers, machinists, and workers in various manufactures requiring the use of lead, therefore, most often suffer. The lead is chiefly taken in by mouth from unclean hands, but may be inspired, especially when men work in poorly ventilated rooms, or may be absorbed from the clothing. Water, cider, or wine confined in lead casks or passed through new lead pipes may poison. Women are sometimes poisoned by hair-dyes or cosmetics containing lead or by biting lead-dyed silk thread.

Morbid Anatomy.—The blue line is found in the margin of the gums of the incisor or canine teeth, especially on the lower jaw. It is due to a deposit of lead sulphide, formed by combination of lead circulating in the blood with the sulphur of hydrogen sulphide developed from decomposing food. Arteriosclerosis, chronic endocarditis, chronic nephritis and gouty lesions may be found. The peripheral nerves show degeneration in cases with nerve lesions and the muscles may be atrophic and degenerated. The brain may show anemia, edema, and degenerative lesions of the cortex.

Symptoms.—These usually develop after protracted exposure, but may present themselves in a few days, if the intoxication is rapid. This is most likely to occur in men inhaling fumes heavily charged with lead from smelters. The symptoms in some cases develop so rapidly as to resemble an acute metallic poisoning; usually they are evolved slowly, and whether of acute or chronic type are of the same general character, including (1) Anemia. A secondary anemia with reduction of red cells and hemoglobin is present. In addition the blood shows the so-called basophilic degeneration of the erythrocytes, the cells showing a fine stippling with granules which take up the basic stains. (2) The blue line on the gums of the incisor and possibly the canine teeth. It is best seen in the lower gums as a fine bluish line in the margin of the gums, not removable by cleansing. (3) Lead palsy. Various nerves may be involved in the neuritis and several forms of lead palsy are described. (a) The wrist-drop or antibrachial type is the most common. When the arms are extended the hands droop and cannot be raised, because of palsy of the extensors of the wrist. The muscles may atrophy. (b) The brachial type, the Duchenne-Erb type, involving the deltoid, biceps, and brachialis anticus, sometimes the supra- and infraspinati. (c) The Aran-Duchenne type, involving the interossei of the hand and the muscles of the thenar and hypothenar eminences. (d) The peroneal type, involving the peronei and extensors of the toes, and producing foot-drop and the steppage gait. (e) A laryngeal type, involving the adductors of the larynx. Lead palsy is regularly bilateral and symmetrical, without sensory symptoms, and accompanied by atrophy. Reaction of degeneration is usually present. (4) Colic. Attacks of abdominal pain due to spasmodic contraction of the intestine are one of the commonest features of lead poisoning. The attacks are mild or

so severe as to produce collapse. The pain is regularly referred to the umbilical region. Vomiting may occur with severe colic, and the abdomen is found retracted and the muscles rigid. Pressure usually relieves, but may intensify the pain. The seizures of colic may be repeated at short intervals for many days, or may not recur for long periods. Dull pain and rigidity of the abdomen may continue between the attacks of colic.

(5) Cerebral symptoms. Encephalopathy. Epileptiform convulsions, delirium or coma may occur in severe cases. Transitory hemiplegia or aphasia has been observed. (6) Arteriosclerosis or chronic nephritis may be pronounced in advanced cases. The urine regularly contains traces of albumin and casts.

Diagnosis.—Exposure plays such an important rôle that an occupation involving the handling of lead in any form should always excite suspicion in a patient suffering from colic or palsy. Confirmation must be sought in the presence of the blue line, the basophile degeneration of the erythrocytes, which is not pathognomonic, but highly suggestive, or the demonstration of lead in the urine by chemical tests.

Prognosis is regularly good. Some very acute cases are fatal, or death may result from protracted poisoning producing arteriosclerosis and chronic nephritis. Paralyses usually disappear under protracted treatment.

Treatment.—Prophylaxis is of the greatest practical importance. Cleanliness of the hands and person is essential. Ventilation of industrial plants, the use of respirators and other hygienic measures have greatly reduced the frequency of lead poisoning. Active treatment is usually called for by colic, constipation or palsy. For intense colic the hypodermic injection of morphine may be necessary. Milder cases may be treated by hot applications to the abdomen. The relief of constipation is essential. For this purpose magnesium sulphate in dram doses is given hourly until the bowels move. Enemata of soap suds or oil may also be required. To eliminate lead from the system potassium iodide is given in doses of five grains thrice daily, and water is drunk freely. The iodide is supposed to act by forming a soluble iodide of lead. For the paralysis electricity, either galvanic or faradic, and massage are employed. Iron, strychnine and arsenic may be given as general tonics or for the anemia.

CHRONIC ARSENIC POISONING

Etiology.—Chronic arsenical poisoning may be caused by the continued administration of arsenic for therapeutic purposes in such diseases as chorea, leukemia and the like. It has been caused by the drinking of beer contaminated with arsenic derived from the sulphuric acid used in manufacturing the glucose entering into the production of the beer. Arsenical poisoning is also attributed to the arsenic present in dye-stuffs and used for coloring wall-papers, garments of various kinds, such as cotton dresses or stockings, playing-cards, etc. The curing of

skins, staining of glass, and the use of Paris green as an insect poison
are all possible sources of poisoning. Arsenic is said to be regularly
consumed by the Styrian peasants for the sake of improving the com-
plexion and bodily vigor.

Morbid Anatomy.—Various lesions of the skin, pigmentation,
keratosis, herpes, or urticaria may be found. A degenerative peripheral
neuritis and granular degeneration of the viscera are regularly present.

Symptoms.—Puffiness of the face, nausea, or vomiting, and head-
ache are the early symptoms of poisoning noted when the drug is given
in excessive doses, and regularly cause the withdrawal of the drug.
Peripheral neuritis and lesions of the skin are the important symptoms
in more protracted cases of poisoning. The neuritis first affects the
legs, later the arms or the body generally. Pain in the affected nerves
is usually present and severe. Paralysis follows, involving especially the
flexors of the foot, producing foot-drop and the steppage gait. In severe
cases power and sensation are completely lost in the affected parts.
The skin lesions are important. Pigmentation of brownish tint re-
sembling that of Addison's disease may develop. Keratosis of the palms
and soles, herpes, urticaria or hyperidrosis may be present.

Course and Prognosis.—If the source of poisoning is discovered
and the process stopped, the prognosis is regularly good. The neuritis is
slow and may be protracted, but recovery follows, possibly after months
of treatment.

Diagnosis.—Arsenical poisoning must be distinguished from other
forms of peripheral neuritis, particularly those due to lead and alcohol.
The arsenical neuritis is marked by sensory disturbances, often severe,
by the early involvement of the legs, and by the absence of limitation
to particular muscle groups characteristic of lead palsy. From alcoholic
neuritis it can be distinguished only by the history of the patient, by
other evidences of chronic alcoholism, or by proving the presence of
arsenic in the urine. In any doubtful case appropriate tests should be
made for this purpose. The source of arsenic must also be sought.

Treatment.—The source of the drug must be found and its taking
stopped. Water is given freely to favor elimination. Otherwise the
treatment is that of peripheral neuritis, rest, with later electricity and
massage. In the early stages anodynes may be required.

FOOD POISONING

Food poisoning may arise in several ways: (1) from poisons in-
herent in the food; some edible stuffs are always poisonous, others only
at certain seasons or under special conditions; (2) from poisons
produced by fermentation or decomposition of the food due to bacterial
action; (3) from infection by pathogenic bacteria or parasites carried
in the food, such as typhoid, tubercle, or cholera bacilli, trichinæ or
the cysticercus. The parasitic infections are discussed elsewhere; (4)
from metallic poisons contaminating foodstuffs or beverages. Arsenical

or lead poisoning may be brought about by food, as already described. Canned foods are generally assumed to occasionally cause poisoning by tin or solder, but there is little evidence to support the supposition. The foods most often the cause of poisoning are fish, meats, milk and its products, especially cheese, and some few vegetables.

Poisons inherent in the food—physiological poisons. Certain fish are known to contain substances poisonous to man. Thus in China and Japan the ovaries and testicles of a fish called fugu contain a deadly poison to which the name fugin is given. Some varieties of fish are always poisonous, the tetradon for example, while the organs of others, such as the roe of sturgeon, pike, or barbel, are poisonous only in the spawning season. Fish which eaten cooked is harmless may be poisonous when consumed raw.

Vegetables are much less likely to be poisonous than the animal foods, but certain vegetable poisons are well known, for example the muscarin of poisonous mushrooms, ergot, which sometimes contaminates rye or other grain, and certain vetches whose seed is used as a substitute for wheat.

Bacterial poisons may be produced in fish, flesh, or milk by bacterial action or pathogenic bacteria may be transmitted in food. The development of poisonous substances in food by bacterial action usually accompanies the processes of decomposition. Thorough cooking destroys such poisons. Toxalbumins resembling those of diphtheria or tetanus have been found in decomposing fish. In other cases the poisons belong to the group of ptomaines. Thus from poisonous mussels Brieger isolated a ptomaine which he called mytilotoxin, and the tyrotoxin of cheese is well known.

The more frequent food poisonings are due to the presence of pathogenic bacteria in the food. The tubercle bacillus, the diphtheria bacillus, and the organisms of scarlet or typhoid fever might all be considered under this heading, but are elsewhere discussed. The organisms here of importance are the bacillus proteus, the bacillus enteridis and a variety of organisms belonging in the group of bacillus coli. These organisms have been found in food products as the result or the attendants of disease in fish or meat-animals or as contaminations of food products in the course of their handling. Poisoning of either type occurs most often from raw or imperfectly cooked food.

Symptoms.—The disturbances induced by the consumption of food products containing toxalbumins or ptomaines produced by bacterial action or pathogenic bacteria themselves may be classified in two groups: 1. Those cases in which the symptoms are those of gastro-intestinal irritation, abdominal pain or cramps, nausea and vomiting, and diarrhea with watery stools or stools containing blood and mucus. Fever may or may not be present, and may be of any degree. The accompanying prostration may be slight or may be so severe as to cause death.

2. Cases in which with or without gastro-intestinal symptoms there are marked evidences of disturbance of the central nervous system, such

as headache, delirium, convulsions or even coma; vertigo and staggering gait; dryness of the throat and difficulty in swallowing; salivation; visual disturbances with imperfect vision, sometimes blindness, dilatation of the pupils, and paralysis of ocular muscles or ptosis; great muscular exhaustion and prostration. Disturbances of sensation of various kinds and loss of power in the extremities have been observed. The disturbances of this class may also be slight or may be extreme and death may follow. Convalescence in either of these groups is often slow and difficult.

Vegetable poisons such as muscarin may also produce symptoms of the same character as those described above; in most cases, however, the intoxication is more slowly developed, the clinical symptoms are of more chronic type and malnutrition is pronounced. 1. Ergotism is due to meal or flour made from grain contaminated by the fungus claviceps purpurea. Two types are recognized: (a) Gangrenous, attributed to sphacelinic acid. The marked feature is malnutrition with gangrene of the fingers, toes, ears or nose. (b) Nervous, attributed to cornutin. Headache and weakness appear early, to be followed by muscular cramps and contractions. The spasms recur at various intervals and last for hours or days. Delirium, melancholia, or insanity may follow.

2. Lathyrism is due to the adulteration of flour with the chick-pea vetch. It is unknown here, but occurs in parts of Europe and Asia. The symptoms resemble those of the convulsive type of ergotism.

3. Pellagra is a poisoning due to eating fermented unripe maize or Indian corn. Many cases have recently been observed in the United States. The symptoms include indigestion, insomnia, salivation and diarrhea and an erythematous eruption followed by marked dryness of the skin and desquamation or the development of furuncles, severe nervous disturbances, such as gradual paralysis of the legs, melancholia, or suicidal mania. Death may result. (See p. 420.)

Diagnosis.—Food poisoning usually presents itself in a group of people who have partaken of food together, or in institutions where the inmates have the same diet. In individual instances the relation of the disturbances to the taking of food, often noted to have been tainted or decomposed, is suggestive. From the clinical symptoms the food may be brought under suspicion. The nature of the poison, whether bacterial or chemical, can be determined only by the careful bacteriological or chemical analyses. In the chronic vegetable intoxications evidence must be sought of the contamination of the food and the results of a change to a healthy diet tried.

Treatment.—In bacterial poisonings the indications are to empty the alimentary tract by purges or by washing out the stomach and irrigating the colon. Stimulants may be required for the prostration, and morphine for the relief of pains or diarrhea. Careful feeding and tonics will be required in convalescence. In acute intoxication with vegetable poisons (muscarin) like measures may be indicated. In the more chronic types mentioned above the chief requirement is a change of diet.

CARBONIC MONOXIDE POISONING
(Illuminating Gas Poisoning)

Poisoning by pure carbon monoxide is too rare to be of practical importance, but this carbon compound is the most dangerous component of illuminating gas, and also of the gases of combustion which rise from furnaces, charcoal braziers, and the electrical furnace.

Etiology.—Accidental poisoning occurs occasionally in those working about furnaces, or the charcoal braziers, or from illuminating gas. The great majority of cases result from the inhalation of illuminating gas accidentally or with suicidal intent.

Morbid Anatomy.—The surface of the body is often marked by irregular areas of a cherry-red staining, and there is much post-mortem discoloration. Bronchitis, bronchopneumonia or lobar pneumonia is usually present. The pia mater and brain are congested, and in cases which have survived five or six days a very remarkable softening of the globus pallidus of the lenticular nucleus is found. Nephritis may be found.

Symptoms.—Headache, mental dulness, dyspnea, cyanosis, and prostration are produced in the milder cases of poisoning. In severer cases the victims are found comatose, the skin and mucous membranes deeply cyanotic, the pupils contracted, the conjunctivæ bloodshot, the pulse rapid, bounding and weak, the respiration rapid and labored. The temperature is often raised to 103°–104° F. The patients may die in a few hours, with rising temperature and failing pulse, or they may gradually recover. The cherry-red staining of irregular areas of the skin is often seen during life, and the drawn blood has the same color. Marked rigidity of the extremities may be present. The fever often continues high, irrespective of complications, such as broncho- or lobar pneumonia, which would account for it. The urine is scanty and contains albumin and casts. Those who recover are liable to suffer for indefinite periods from vague nervous disturbances.

Prognosis is always grave, but recoveries occur even after coma lasting for days. In protracted cases broncho- or lobar pneumonia usually develops.

Treatment.—Fresh air is the first requirement and may be sufficient to revive the mild cases. For the severer cases free bleeding with the subsequent intravenous injection of normal salt solution 1000 c.c. is helpful. Oxygen may be administered. Direct transfusion is indicated and may succeed in desperate cases. The reactions of the two bloods should be first tested to be sure that hemolysis does not take place on their mixture. Cardiac and respiratory stimulants are usually required. Feeding must often be carried out by nutrient enemata or by the nasal tube.

SUNSTROKE

(Insolation. Thermic Fever. Siriasis)

Definition.—A morbid condition induced by exposure to extreme heat.

Etiology.—(1) Exposure to high temperatures of any kind may produce prostration. The heat of the sun's rays is the most common cause and produces the severest type of sunstroke, but similar conditions result from high temperatures in-doors, as in boiler-rooms, about furnaces, or in bakeries. (2) Predisposing causes include especially the excessive use of alcohol, and any cause for debility. (3) The loss of sleep incident to protracted hot weather, and hard labor are also important influences.

Morbid Anatomy.—There is a general venous congestion of the skin and internal organs and small hemorrhages may be found in the skin or serous membranes. The brain is congested and under the microscope degenerative lesions resembling those produced by intoxications with alcohol, lead, etc., are found. These are the most characteristic lesions.

Symptoms.—These vary greatly under different conditions and much confusion exists as to the terminology applied to different clinical conditions. The subject will be best understood by considering that the conditions described result from exposure to heat, either of the sun or from other sources, that they are doubtless degrees in one process and that anomalous cases, or cases not falling easily into any group of the classification may be seen.

(1) HEAT EXHAUSTION.—This condition is seen most often in those working in-doors under high temperatures, firemen, bakers, laundresses or cooks and the like. The patient is prostrated, the surface usually pale and clammy, the pulse soft and of normal rate or but little quickened. The mind is perfectly clear, though headache may be complained of. The temperature is normal or subnormal.

(2) SUNSTROKE.—This is commonly the result of working in the direct rays of the sun, but not always. Premonitory symptoms of headache, dizziness, dryness of the skin may be present, or the victim may fall unconscious without warning.

In the milder cases prostration is marked, the temperature is raised to a varying degree, 101° to 105° F., the pulse is rapid and feeble. The skin is cool and moist. There are headache, vertigo, and possibly mild delirium, but not coma. Nausea or vomiting may occur. Recovery is the rule.

In the severe cases the patient passes suddenly into coma, the face and skin generally are flushed, dry, and burning hot, the pupils are normal or contracted, the extremities are sometimes rigid, sometimes flaccid, not infrequently convulsions occur, and urine and feces are discharged involuntarily. The temperature ranges from 105° to 115° F., the pulse is very rapid, and correspondingly weak, the respiration is

rapid and noisy, the lips and finger-nails are cyanotic. Unless relieved by prompt treatment the patient dies in a few hours. In those recovering under treatment the temperature falls rapidly, consciousness returns in part at least, the pulse improves and the patient gradually returns to a normal condition. Secondary rises of temperature with stupor or coma and other manifestations of the primary stroke may occur. Coma may last for days.

Diagnosis.—The conditions under which the prostration occurs usually render diagnosis easy. The absence of other causes for prostration, such as heart failure, is important. In the febrile cases severe malarial paroxysms may be overlooked. The enlarged spleen and the examination of the blood should prevent error.

Prognosis.—The milder cases recover. Of the severer ones, those with temperatures of 105° or over, the mortality will reach 10 per cent.

Treatment.—For heat exhaustion and the milder cases of sunstroke, rest in a cool and quiet room, with a sponge bath if the temperature be high, and moderate stimulation by whiskey or brandy, is all that is required. For severe sunstroke the patient is stripped and either rubbed with cakes of ice or put in an iced bath. A thermometer is kept in the rectum and when the temperature has fallen to 105° or 104° the rubbing or bath is stopped. The temperature continues to fall to normal or even below. Camphor and ether, caffeine, strychnine, or other cardiac stimulants are given hypodermatically, if the pulse fails. The temperature must be watched and a cold bath given, if it begins to rise again. When these methods cannot be carried out, douching the body with cold water may be tried.

DISEASES OF THE MUSCLES

MYOSITIS

Myositis occurs in some forms of muscular rheumatism, in trichiniasis, and in pyemia, or in localized abscesses. Otherwise myositis is a rare affection. It has, however, been encountered and described under several different forms.

(1) PRIMARY SUPPURATIVE MYOSITIS.—This affection is seen frequently in Japan. There are multiple localized areas of induration in the muscles, and abscesses are often found. Pyogenic bacteria are found in the abscesses. The constitutional symptoms are those of pyemic infection, and the possibility of a cryptogenic pyemia can hardly be excluded.

(2) DERMATOMYOSITIS.—This is an acute, sub-acute or chronic disease, of unknown etiology, characterized by a gradual onset of localized areas of edema, dermatitis and myositis. The morbid anatomy consists in a definite myositis with swelling of the muscle, edema, infiltration with leukocytes and degeneration of the muscle fibers. Hemorrhages may be found in the muscles.

Symptoms.—Vague pains throughout the body mark the onset, later the pains become so severe that the patient is bed-ridden. Fever, perspiration, and enlargement of the spleen are observed. Then areas of edema and dermatitis overlying the inflamed muscle develop on various non-symmetrical parts of the body. Stomatitis and angina develop in some cases and involvement of the muscles of deglutition and respiration often lead to death from suffocation or bronchopneumonia.

The course of the disease may be very acute, one to two weeks, or very chronic, as many years. More than half the reported cases resulted fatally.

Treatment must be directed to the relief of pain and the maintenance of nutrition.

MYOSITIS OSSIFICANS.—This is a very rare disease, seen occasionally in boys at puberty. Areas of myositis develop which after a time subside, leaving areas of connective tissue formation in which cartilage and bone are ultimately deposited. The process therefore results in gradual deposition of these substances in various muscles throughout the body, leading in the end to great disability and pseudo-ankylosis of joints. The temporals, pterygoids and masseters are finally involved and the patient is unable to open his mouth or masticate his food. Exostoses are common. The disease lasts for many years. The subjects die from intercurrent infections. Many medicines have been used without result. Operative measures may be necessary.

MYOTONIA CONGENITA

(Thomsen's Disease)

Definition.—A congenital nervous affection characterized by muscular cramps at the beginning of voluntary movements.

Etiology.—The disease occurs in certain families. Consanguinity is thought to be a factor. Fright or injury sometimes precedes the development of symptoms. Males are more often affected.

Morbid Anatomy.—Sections of muscle examined during life have shown an increase in the nuclei in the muscle fibers, an increased width of the fibers and various modifications of their protoplasm. The latter may show vacuolation, disappearance of normal striation, fibrillation, or other degenerative changes. These changes are probably the results rather than the causes of the disease.

Symptoms.—These commonly develop above the age of 20, but may appear during childhood or puberty. Military service has first brought them out in a number of cases. Tonic cramps, without pain, develop in the muscles on attempting any voluntary movement. The cramps recur several times on the repetition of the effort and then vanish, so that the muscles can be used normally. The result is a certain awkwardness in the performance of any movement till the cramps have passed off. All the voluntary muscles may be affected, and the difficulty appears in walking, shaking hands, speaking or eating. In some instances

a single group of muscles is involved. The muscular contractions are slow, weak, and last some time after the effort which excites them. The muscles become hypertrophied and the patient looks strong, but is actually below normal in power. The persistence of the contractions after electrical stimulation is the chief feature of these reactions. Tendon reflexes are normal, deficient or increased.

The disease varies from mild to severe grades, is chronic and incurable. No effective treatment has been found.

MYASTHENIA GRAVIS

(Asthenic Bulbar Paralysis)

Definition.—A disease characterized by signs of extreme muscular fatigue, without definite lesions of either nervous or muscular system.

Etiology.—The disease commonly develops between the ages of twenty and thirty, but has been observed in infancy and old age.

Previous acute disease, menstruation, pregnancy, and the menopause apparently bring out the symptoms, but can hardly be regarded as causing the disease.

Morbid Anatomy.—The nervous system is normal; the muscles may show an infiltration of lymphocytes between the muscle fibers.

Symptoms.—The onset is slow, insidious. The evidences of the disease usually appear first in the muscles of the eyes, the jaws or lips. Ptosis, diplopia, and difficulty in mastication are therefore early symptoms. The ptosis or other symptoms disappear after rest or sleep, to recur on any effort and finally become permanent. All the muscles of the body may finally be affected. The muscles of respiration are involved in the terminal stage, causing dyspnea and ending in bronchopneumonia.

MYASTHENIC REACTION.—A faradic current applied to affected muscle excites a strong contraction, which, however, soon lessens, till finally there is no response. The same early fatigue can be demonstrated by the attempt voluntarily to repeat a given muscular contraction, such as closing the eye, chewing or whistling. The muscles quickly fail and can only be made to act again after rest. The reflexes are exaggerated. Sensory symptoms are lacking. The diagnosis is easy, if the symptoms are borne in mind and the muscles tested by electricity and voluntary action. The disease in its early stages is often mistaken for hysteria.

Many cases result fatally; recovery is possible. Prolonged rest with systemic tonics constitute the treatment.

PARAMYOCLONUS MULTIPLEX

A nervous affection characterized by clonic lightning-like contractions of the muscles, usually those of the lower extremities. The etiology and pathology are obscure. The affected muscles, the vastus, rectus, or adductors, or, in the arm, the biceps, triceps, or supinator longus, con-

tract swiftly as though electrically stimulated, but, as a rule, no movement results.

The electrical reactions are normal. The contractions may be increased by mechanical irritation of the skin or the muscles themselves. Treatment must be directed to the general condition of the patient.

RARE DISEASES OF THE BONES AND JOINTS

(Osteitis Deformans, or Paget's Disease)

Definition.—A rare chronic disease of the skeleton characterized by enlargement of the cranial vault and forward projection of the head with dorso-cervical kyphosis, prominence of the clavicles, widening of the base of the thorax, a relative increase in the width of the hips and a forward bowing of the legs. The etiology is unknown. It appears sometimes to be closely associated with the growth of malignant tumors, carcinoma or sarcoma.

The morbid anatomy combines hypertrophic and rarefying osteitis.

The symptoms consist of the bony changes and irregular shifting pains, possibly headache. The shortening of stature from bowing of the spine and legs may amount to a foot or more. The disease is progressive, but the patients die of intercurrent infections.

LEONTIASIS OSSEA is perhaps a variety of Paget's disease affecting particularly the bones of the head and face. The steady increase in the size of the head is the striking feature of the affection. Headache, insomnia, apathy, mental dulness, or epilepsy may be associated with the bony changes. The condition may later develop into Paget's disease.

HYPERTROPHIC PULMONARY OSTEOARTHROPATHY is an enlargement of the bones and to some extent of the soft tissues of the extremities, especially the fingers and toes. The enlargement is symmetrical. (See Fig. 1.) In the fingers and toes the increased size is due to hypertrophy of the soft parts. In the arm or leg there may be marked thickening of the distal ends of the long bones. Occasionally effusions into the joints are found. The condition is closely associated with chronic pulmonary disease, especially bronchiectasis, but is often seen in congenital heart disease and a variety of other affections.

VIII.

THE INFECTIOUS DISEASES

TYPHOID FEVER

(Enteric Fever. Typhus Abdominalis)

Definition.—Typhoid fever is an acute general infection due to a specific micro-organism—the *bacillus typhosus*—marked by characteristic lesions in the intestinal lymph follicles and elsewhere, and manifested clinically by protracted fever, prostration, intestinal disturbances, enlargement of the spleen and a rose-colored skin eruption.

Etiology.—Typhoid fever is one of the most widely distributed of the infectious diseases. Although its greatest prevalence is in temperate regions it seems little affected by climate or altitude, nor do races show much difference in susceptibility. It is more common in cities than in the country and occurs both endemically and in small, local epidemics. Its prevalence in a community bears a direct relation usually to conditions of sanitation, and especially to the purity of the water supply.

SEASON.—The disease prevails chiefly in the late summer and autumn. Considerably more than half of the cases occur during the months of August, September, October and November. A hot, dry summer seems to have some influence in augmenting the number of cases.

AGE.—Typhoid fever is chiefly a disease of young adult life. Liebermeister's statistics showed that 58 per cent. of the cases occurred between the age of 20 and 30 years. It is not uncommon in older children and in middle life, but cases are extremely rare in infants under one year and in old age.

SEX.—Males seem slightly more liable to attack than females.

CONDITION OF HEALTH.—It has long been noticed that the disease is especially prone to attack the vigorous and well-nourished individuals rather than the weakly. The well-to-do suffer equally with the poor. Pregnancy, the puerperal state, lactation and certain of the infectious diseases seem rather to diminish susceptibility to the disease (Curschmann).

IMMUNITY.—Variations in individual susceptibility doubtless exist. One attack usually confers immunity, but two and even three attacks in the same person have been observed.

BACTERIOLOGY.—The direct, exciting cause of typhoid fever is a specific micro-organism—the bacillus typhosus, discovered in 1880 by Eberth and carefully studied by Gaffky and others. It is a short, rather thick, flagellated, actively motile rod whose length is one-third of the diameter of a red cell. It grows readily on most of the usual culture media and does not form spores.

In the body the bacilli are found abundantly in the intestinal lesions, the mesenteric lymph-nodes, the spleen and, less constantly, in the bone marrow, liver, gall-bladder and other organs. Recently, by improved

methods, they have been isolated from the circulating blood in about three-fourths of all the cases examined. They have also frequently been demonstrated in the rose spots. In the stools of patients the germs are found almost constantly in great abundance. In a certain proportion of cases (20–30 per cent.), moreover, they are found abundantly in the urine. These are the two chief avenues by which the bacilli leave the body and by which transmission of the disease occurs. The sputum rarely contains the bacteria and the sweat and expired air probably never.

Outside of the body typhoid bacilli may, under even moderately favorable conditions, live and remain active for weeks and months. They are readily destroyed by even weak solutions of such germicides as carbolic acid and the bichloride of mercury and by exposure for fifteen minutes to a temperature of 60° C. On the other hand, they are very tolerant of cold and may retain their vitality when kept in ice for as long as three months.

Exposure to direct sunlight will destroy them in a few hours. They are known to live in fecal matter, water, earth, and even dust, for at least many weeks. Whether under such circumstances they are capable of multiplication is still uncertain.

Transmission.—Typhoid fever may be contagious in the usual meaning of the term. That the germs enter the body, in the vast majority of cases, through the alimentary tract cannot be doubted. Indeed, evidence is lacking that infection ever occurs by any other channel.

Contaminated drinking water is by far the most important medium of infection. Streams, reservoirs, wells, springs, exposed to pollution from typhoid discharges by means of surface drainage or from neighboring cesspools, are the usual sources.

A number of epidemics have been traced to milk, infected by contaminated water. Oysters fattened at the mouth of polluted streams have been shown to be the source of at least several small epidemics. The transference of infectious material by flies and other insects deserves greater attention than it has hitherto received. Where cesspools are exposed and open, as in the country districts and especially in army camps, this means of infection is probably often an important one. Nurses and attendants upon typhoid patients occasionally infect themselves through lack of care in handling bedpans, soiled linen, etc.

Of recent years typhoid bacilli have been repeatedly found in the feces of persons who had had the fever months or years before, and in some instances in those who had never had the fever, and localized epidemics have been traced to direct infection from such '' carriers '' employed as cooks or the like.

Morbid Anatomy.—The essential lesions of typhoid fever are found in the lymph tissue of the intestines, the mesenteric lymph-nodes and the spleen.

Intestines.—The lymph tissue of the intestines is made up of solitary follicles, which are scattered widely through small intestine, vermiform appendix and colon; and Peyer's patches, which are aggre-

gations of solitary follicles occurring chiefly in the ileum. Although these structures are liable to involvement everywhere throughout the length of the intestines the lesions are regularly most numerous and extensive in the lower part of the ileum, where the lymphoid tissue is most abundant.

The changes taking place in both the Peyer's patches and the solitary follicles are identical in character and may conveniently be divided into several stages.

(1) Congestion and hyperplasia.

(2) Necrosis and sloughing.

(3) Ulceration.

(4) Cicatrization.

1. *Congestion and Hyperplasia.*—The glands affected are of reddish gray color and are swollen and somewhat raised above the surrounding mucous surface; the solitary follicles varying in size from that of small shot to that of a pea. Microscopically there is at first hyperemia followed by a great increase of the lymphoid cells of the gland ('' medullary infiltration ''). As this hyperemia increases the blood-vessels are compressed and the patch becomes grayish white in color. Early in the second week of the disease the hyperplasia of the gland usually reaches its height and now involution may occur in one of two ways. In the milder cases the accumulated cells of most of the glands undergo degeneration and absorption and the gland soon regains its normal state. More commonly, however, most of the glands undergo—

(2) *Necrosis and Sloughing.*—This necrosis is due in part to the compression of the blood-vessels (anemic necrosis), and in part, probably, to the action of the toxins of the typhoid bacilli. The necrosis may involve only a small part of a gland, or the entire surface of a patch may die and form a slough.

(3) *Ulceration.*—From the separation of these sloughs there result the *ulcers* which are the most conspicuous feature of the typhoid lesions. They correspond in position, of course, to the affected Peyer's patches and solitary follicles, but are by no means always co-extensive with the limits of these glands. Usually, indeed, they occupy only a portion of the surface of such glands. Their outlines may be round, oval or quite irregular. Their edges are usually sharp and clean cut. In depth they vary exceedingly. Some are so shallow as to involve only the superficial layers of the mucosa. Usually they extend to the submucous or muscular layer, occasionally they penetrate to the peritoneal coat or even perforate the entire wall. This latter event with its consequent septic peritonitis constitutes one of the gravest of the complications of the disease. Moreover, the separation of the sloughs and the development of the ulcers often result in the erosion of blood-vessels and free hemorrhage into the intestine. Ulceration is usually most extensive in that part of the ileum immediately above the ileo-cecal valve, as it is here that Peyer's patches are largest and most numerous. The stage of ulceration occupies usually the latter half of the second and third weeks of the illness.

(4) *Cicatrization.*—In ordinary cases the process of healing begins almost as soon as the ulcers are well developed. The loss of substance is gradually filled up by granulation tissue, the epithelium extends over from the edge of the ulcer and a scar results, which in the course of time becomes somewhat depressed and often pigmented. Such scars, however, rarely produce serious constriction of the bowel. The healing process is usually well under way by the beginning of the fourth week. Sometimes, however, when the recuperative power is small, healing may be delayed for many days.

In addition to the specific lesions mentioned, both small and large intestine are usually the seat of more or less extensive catarrhal inflammation to which, rather than to the change in the lymph follicles, are attributed certain of the intestinal symptoms and in particular the characteristic ''pea-soup'' stools. The ulceration may extend into the colon and even become extensive there.

Typhoid Fever Without Intestinal Lesions.—The severity of the symptoms by no means always corresponds with that of the intestinal lesions. In some cases these latter may be very slight and inconspicuous. Indeed, in a few fatal cases recently reported no characteristic intestinal changes whatever could be found, although typhoid bacilli were isolated from the blood and organs.

The Mesenteric Lymph-Nodes.—The same process of lymphoid hyperplasia is seen in the lymph-nodes of the mesentery, especially in those of that part of the mesentery which is opposite to the site of the chief intestinal lesions. The nodes are much swollen and may be seen in the mesentery at autopsy as nodules, often as large as a cherry. In these extensive necrosis is much less common than in the intestinal follicles, although small foci of necrosis are not infrequent. Usually the medullary infiltration undergoes absorption and resolution and the nodes return to normal with the subsidence of the intestinal lesions. If, as happens occasionally, these swollen nodes are secondarily infected by pyogenic organisms, suppuration and abscess may result. Even the retroperitoneal nodes may share in the swelling and hyperplasia.

The Spleen.—The enlargement of the spleen occurs early and constantly. At autopsy it is found usually to be from $1\frac{1}{2}$ to 3 times its normal size. This increase depends upon its engorgement with blood and upon the marked hyperplasia of its lymphoid elements. It is usually dark red or brownish in color and soft or even diffluent in consistency. Occasionally it is the seat of infarct or abscess. Spontaneous rupture has occurred.

Other Lesions.—*The liver* shows constantly a greater or less degree of degeneration, albuminous or fatty, of the hepatic cells. Frequently also minute foci of lymphoid cells are found scattered through it which may undergo necrosis. Liver abscess is a somewhat rare complication.

Inflammation of the *gall-bladder* and *large bile-ducts* is not uncommon.

Heart.—The myocardium is frequently involved and shows two types of lesions—(1) a degeneration of the muscle fibers, *parenchymatous de-*

generation; and (2) an exudative inflammation of the interstitial connective tissue—*acute myocarditis.* *Endocarditis* and *pericarditis* are rare.

BLOOD-VESSELS.—Thrombosis of an artery with consequent gangrene of the part is rarely seen. A similar thrombosis of the large veins, especially of the femoral, is less uncommon. It depends upon an inflammation of the vein wall (phlebitis) caused, in some cases at least, by the typhoid bacilli themselves.

The larynx is very commonly the seat of a catarrhal laryngitis. Occasionally a severer type of inflammation with ulceration and even with destruction of the cartilages is found.

LUNGS.—In the lungs a great variety of lesions occur. Lobar and lobular pneumonia, gangrene, abscess and hemorrhagic infarction are all occasionally seen. The rôle of the typhoid bacilli in these processes is still unsettled. Frequently, at least, they are due to infection by other bacteria. Hypostatic congestion is often found in the fatal cases. Fibrinous or serous *pleurisy* is not of very frequent occurrence. In some of these cases, and in the rare cases of empyema, typhoid bacilli in pure culture have been found.

KIDNEYS.—Parenchymatous degeneration of varying degree is the rule and in fatal cases true acute nephritis is not infrequently met with. Catarrhal and suppurative inflammations of the pelvis of the kidney and of the bladder, due to the bacillus typhosus, occur.

Among the rare lesions found are acute leptomeningitis, parotitis, periostitis, osteomyelitis and joint inflammations. The voluntary muscles, especially those of the abdomen, show not infrequently a peculiar type of degeneration which sometimes results in hemorrhage or spontaneous rupture.

Symptomatology.—It will be convenient to give first a general description of the symptoms and course of the disease in its typical form and in the common variations from this, and to take up later in detail the individual symptoms and complications.

PERIOD OF INCUBATION.—In infectious diseases the time elapsing between the entrance of the specific germs and the manifestations of the first definite symptoms is known as the period of incubation. In typhoid fever this period is less easy to estimate accurately than in the case of contagious diseases such as the exanthemata. Usually, however, it varies between one and two weeks. Occasionally it is as much as three weeks and it may be even longer. This period is often quite free from symptoms, but not infrequently there are noticed vague discomfort, languor and failing appetite.

COURSE AND GENERAL DESCRIPTION.—The acute stage of the disease commonly lasts from three to five weeks, the first day of fever usually being counted the first day of the disease.

Onset.—First Week.—The onset is almost always insidious and gradual. Lassitude, loss of appetite, headache, pain in the back and legs, fever and chilliness are the symptoms usually complained of. A distinct rigor is uncommon. The headache is usually frontal and is often severe

and persistent. It is, however, by no means always present. Epistaxis is common. The face is flushed; the tongue usually small, moist, coated with a yellow or whitish fur and often clean and red at the edges and tip. There may be nausea and vomiting. Constipation is the rule, but mild diarrhea may be present from the beginning. The abdomen is soft and tenderness is slight or lacking. The fever usually rises from day to day and at the end of the first week may reach 104°–105° F. The pulse is slightly accelerated (80–100), full, of low tension and sometimes dicrotic. A mild grade of pharyngitis, laryngitis or bronchitis is of very common occurrence.

Second Week.—The fever reaches its height usually before the beginning of the second week and continues high throughout the week. All the evidences of continued fever are now marked. The tongue and lips are dry, the face is flushed but heavy and apathetic. The patient is apt to sleep most of the time, but the sleep is restless and troubled. If delirium be present it is usually of the quiet, muttering type. The pulse is somewhat more rapid and less strong. At the beginning of this week or at the end of the first week two symptoms of great diagnostic importance usually appear: (a) the eruption of rose-spots upon the abdomen (7th to 9th day) and (b) the distinct enlargement of the spleen. Tympanites and diarrhea are common. Intestinal hemorrhage may occur. Death may result during this week from severe nervous disturbance or from early complications.

Third Week.—In mild cases this week may mark the beginning of convalescence. In the average cases, however, it is the critical period of the disease; the time when intestinal ulceration is most extensive, when the danger of hemorrhage and perforation is greatest and when complications, especially pulmonary ones, are most likely to develop. The temperature still continues high, although less uniformly so. The patient lies constantly upon his back, completely prostrated, anemic and emaciated. The tongue is dry and cracked, the teeth and lips covered with sordes. Tympanites and diarrhea are common. Mental apathy is extreme. There is apt to be stupor or quiet delirium. The heart action is rapid and feeble, the skin pale and the extremities often slightly cyanotic. Toward the end of the week, in favorable cases, the temperature begins to fall and the evidences of toxemia become less marked.

In the *fourth week*, in such cases, the temperature gradually falls to normal, the patient grows brighter and the heart action stronger. The tongue again becomes moist and the appetite returns, but prostration and emaciation are still marked. In protracted cases the features of the third week are continued into the fourth or even into the eighth or ninth week of the disease.

Convalescence, even if uninterrupted by complications or relapses, is slow and tedious. The regaining of the lost flesh (often 30 or 40 pounds) requires usually several weeks at least and the return of strength is no more rapid.

Variations.—1. In Mode of Onset.—Instead of the usual insidious onset the disease may begin in one of the following ways:

(a) Suddenly with one or more *chills* and with an abrupt rise of temperature.

(b) *With persistent and uncontrollable vomiting,* either with or without severe diarrhea.

(c) *With pronounced pulmonary symptoms.* These are most commonly symptoms of a severe and general bronchitis. Occasionally, however, the disease begins with the symptoms and signs of a lobar pneumonia and its true nature may not be evident for some days—the so-called ''pneumo-typhoid.''

(d) With severe *nervous* symptoms. Intense headache, insomnia, neuralgic pains, photophobia, delirium, even retraction of the head and convulsions, may be present in varying combinations. The symptoms may for some days closely simulate meningitis.

(e) With symptoms of an acute nephritis—''nephrotyphoid.''

2. I n C o u r s e.—Departures from the course above described are numerous and varied. There are,

(a) Mild cases, in which all the symptoms, including the temperature, are relatively slight and in which the course is usually short (ten days to three weeks).

(b) Protracted cases usually, but not always, severe, in which the fever and the other symptoms, even when uncomplicated, may continue for five, six or even nine weeks.

(c) Malignant or Fulminant Typhoid.—In which from beginning the infection is very intense, the temperature very high and the heart action rapid and feeble. Such cases may be fatal even in the second week.

(d) Abortive Typhoid.—In which after the usual onset, or one more abrupt than usual, the disease is interrupted at the beginning, middle or end of the second week by a rapid, or even critical, defervescence and a speedy recovery. Such cases are by no means always mild ones.

(e) Ambulatory Typhoid (''Walking Typhoid'').—An important and not uncommon class of cases in which during the early stage the patient has not felt sick enough to go to bed, but has kept about and perhaps at work. Such cases often first come under observation during the second week or even later. They may apply for treatment only when intestinal hemorrhage or perforation has occurred.

(f) Hemorrhagic Typhoid.—Rare cases, often fatal, marked by development of purpuric symptoms such as hemorrhage beneath the skin, from the mucous membrane and into the viscera.

I n C h i l d r e n.—The disease in children is apt to run a somewhat shorter and milder course than in adults. The fever is often of a more distinctly remittent type, prostration is less pronounced and the severe intestinal complications of hemorrhage and perforation less common. On the other hand, relapses are of distinctly greater frequency. The mortality is very low.

I n t h e A g e d.—Typhoid fever is marked by a tendency to early and great prostration, cardiac depression, delirium and fatal pulmonary complications.

Afebrile Typhoid.—Cases of typhoid infection *without fever* occur, but are extremely rare. Convalescent patients may show bacilli in the feces for years. Occasionally the bacilli are found in those who have never had the fever.

INDIVIDUAL SYMPTOMS.—*Fever.*—The course of the fever may be conveniently divided into three stages: the initial rise, the period of febrile elevation or fastigium, and the period of defervescence. *The initial rise* occupies from four to six days. There is usually a step-like increase from day to day, the fever each evening being one or one and a half degrees higher than that of the evening before. This same increase is to be seen also in the morning temperature, which, however, is regularly from one and a half to two degrees lower than that of the evening of the same day. This initial rise is sometimes much more abrupt and may occupy only two days or even less. (See Fig. 61.)
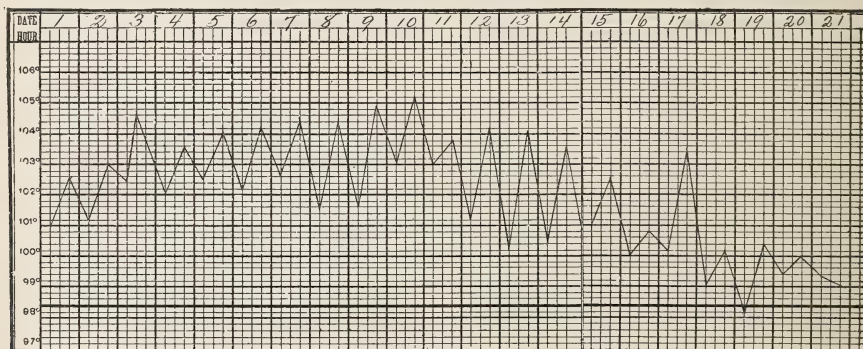
FIG. 61.—Temperature of typhoid fever, showing the gradual rise, the fastigium or height of the fever, and the fall by lysis. A normal course of three weeks.

The fastigium or stage of febrile elevation occupies, in typical cases, the second and third weeks of the disease. During this time the fever maintains a fairly constant elevation varying from 103° to 105° F., the morning remissions being gradually not more than one degree. In the milder cases the temperature may reach only 102° or 102½°. Occasionally in severe types it rises to 106° or even more. The duration of this stage varies greatly. In many of the cases it is not longer than one week. In the protracted cases it may be three, four or even six weeks in extent. In the latter part of this stage the morning remissions are generally increased to two or even three degrees, while the evening temperature still maintains its height. The increased drop in the morning temperature is usually the first sign of beginning defervescence. Sudden, violent drops in temperature are uncommon except as the result of severe intestinal hemorrhage, perforation or profuse sweating. Occasionally, however, the fever may be markedly remittent throughout.

The stage of defervescence is usually of about one week's duration. During this time the temperature gradually falls to

normal, the evening rise being the last to disappear. Following this there are usually a few days of slightly sub-normal temperature. Many variations from the course described are seen in the stage of defervescence. The period may be much shortened, or much prolonged, or the daily step-like fall may be variously disturbed.

Fever During Convalescence.—This may be (a) that of the so-called recrudescence—a transient and sudden rise, attributable usually to constipation, to indiscretion in diet or to some excitement; (b) that of some complication, and (c) that of a true relapse.

Relapses.—These are met with in about 10 per cent. of all cases. They are believed to be always a true reinfection by typhoid bacilli remaining in the body (e.g., in the gall-bladder). The fever shows the same gradual rise and fall as that of the original attack, although its course is usually shorter (two to three weeks). (See Fig. 62.) All the other symptoms, including the rose-rash and the enlargement of the
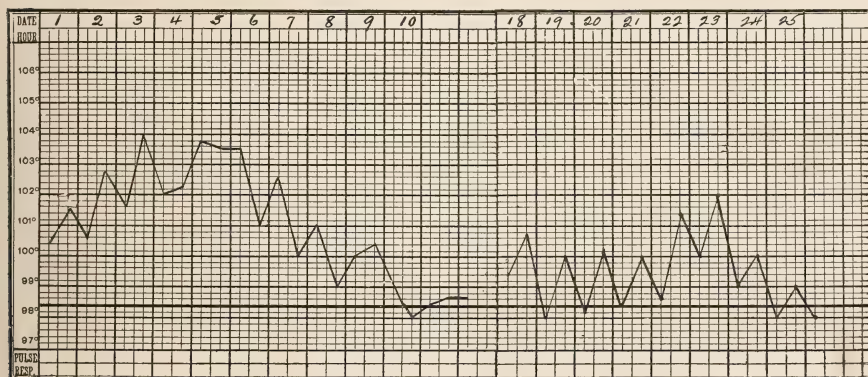
Fig. 62.—Mild typhoid of 10 days' duration. Temperature normal for one week after this, followed by irregular fever as in Chart II. Widal negative, blood culture sterile during this period. Widal positive, blood culture positive first time on the 21st day of the disease.

spleen, are usually seen. The relapse begins in most cases a few days (5 to 14) after defervescence is complete, but the internal may be as as great as forty or fifty days. On the other hand, it may begin during defervescence and before the temperature has reached the normal ("intercurrent relapse"). A second or even a third relapse is not very uncommon.

These relapses should be carefully distinguished from the other forms of post-typhoid fever just mentioned.

Chills, while not usual in typhoid, are occasionally seen. They may mark the onset of the disease or the development of some complication. At other times they occur without known cause.

Pulse.—The pulse is regularly increased in rate, but, in the early stage at least, only very moderately. During the first few days it is usually below 100 in rate and is full and tense. It soon becomes softer, however, and then, frequently, *dicrotic*. As the disease progresses and

exhaustion increases the rate gradually rises to 120, 130 or even more. At the same time the pulse becomes smaller and softer and the dicrotism disappears. Irregularity in force or rhythm is not common and is apt to be of serious import. During convalescence the pulse rate is easily disturbed by slight exertion or excitement. Bradycardia is occasionally seen at this time.

Digestive Symptoms.—T o n g u e.—The appearance of the tongue at the outset has been described. Later it often becomes a sign of some prognostic value. In the mild and moderate cases which are doing well it remains moist throughout. In severe cases it becomes very dry and tremulous and is often covered by hard, brownish crusts. The gradual return of moisture to the tongue is one of the early evidences of improvement.

The appetite is generally lost at the onset and remains so until the advent of convalescence. Nausea and vomiting are not common symptoms, but occasionally they may be so persistent and unmanageable as to be a serious menace to life.

D i a r r h e a is a symptom of much less frequence and importance than is popularly supposed. It is present throughout the whole or greater part of the attack in rather less than one-third of the cases. In another third, approximately, the bowels are constipated throughout, or the movements are fairly normal. When present the diarrhea is usually not severe and is free from pain and tenesmus. It seems to depend rather upon the associated catarrhal enteritis than the specific typhoid lesions and bears little or no relation to the severity of these latter. Such stools are usually copious, thin, fecal and of light yellow or grayish color—the so-called ''pea-soup'' stools. Exceptionally the diarrhea may be severe and uncontrollable and thus form a conspicuous and serious symptom.

T y m p a n i t e s.—A moderate degree of flatulence is commonly seen and has little significance. Occasionally, however, the intestinal distention becomes very great and persistent and then may cause serious disturbance of the cardiac and respiratory functions. It may also excite vomiting or persistent hiccough, and it no doubt increases the liability to perforation.

Slight *tenderness*, especially in the right lower quadrant of the abdomen, is frequently met with. Occasionally this may be so marked as to suggest appendicitis. Abdominal *pain* is also sometimes felt, but is rarely severe except when due to some complication such as perforation.

The *gurgling* so frequently felt on pressure in the right iliac fossa has little diagnostic value.

Intestinal *hemorrhage* and *perforation* will be discussed under complications.

Eruption.—The characteristic rash of typhoid consists of rose-red spots or papules somewhat larger than the head of a pin, of velvety feel and only slightly elevated. They appear usually about the seventh

or eighth day of the disease and are scattered sparsely over abdomen, chest, back and, less frequently, extremities (see Fig. 63). Occasionally they become very numerous over the trunk and extremities, but they always remain discrete. Each spot consists of a hyperemic (not hemorrhagic) area which completely disappears upon pressure. They appear in successive small crops, each spot persisting from three to five days, while the whole eruption lasts from ten to twelve days. In some cases the entire eruption will consist of not more than half a dozen spots; moreover, the eruption fails altogether in 15 or 20 per cent. of the cases. Recently typhoid bacilli have been found in a large portion of the rose spots examined and it seems probable that they always contain such bacilli.

Spleen.—Moderate enlargement of the spleen is almost invariable and begins early. By the sixth or seventh day its tip can usually be felt well below the edge of the ribs. Both palpation and percussion
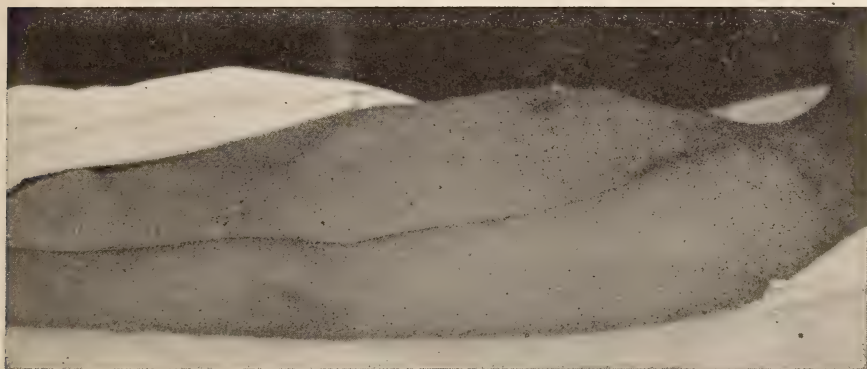
Fig. 63.—Typhoid fever with profuse eruption, spots plentiful on abdomen, chest and arms.

may, however, fail to discover it if there be much tympanites or abdominal rigidity. Slight tenderness is not uncommon. The enlargement subsides usually with the abatement of the fever.

Cerebral Symptoms.—H e a d a c h e, during the first week, is rarely altogether lacking. Usually it is a constant dull, frontal or occipital pain. Occasionally it takes the form of an intense occipital, temporal or facial neuralgia. Sleeplessness, restlessness and mild photophobia are common during this period. Occasionally all these symptoms may be very marked and to them may be added delirium, rigidity of the neck, muscular twitchings, etc., so that the picture may closely simulate that of meningitis.

In the second week the headache and restlessness usually give place to drowsiness and mental apathy. The discomforts of the illness are less appreciated by the patient, his interest in his surroundings flags and he lies most of the time in a drowsy, stuporous condition and shows distinct aversion to every sort of mental effort. *Delirium* is present at

some time in most, but by no means in all, of the cases. Often there is merely a slight wandering of the mind at night. The delirium is rarely an active, violent one. The possibility that a delirious patient may leave his bed, leap from a window or otherwise harm himself must always be borne in mind. Usually the patient lies quietly, with open eyes and muttering speech, oblivious to his surroundings yet easily aroused. As the prostration increases there are added tremor of the hands and lips, involuntary twitching of the muscles of the arms and legs (subsultus tendinum), and automatic movements of the hands, such as picking at the bed clothes. Feces and urine may be passed involuntarily or, what is more common, retention of urine may occur and require catheterization.

Blood.—The red cells show a gradual diminution in number and in the percentage of hemoglobin, which is usually only moderate and which develops chiefly in the latter part of the disease. The condition of the *leukocytes* is of more importance. In contradistinction to most acute diseases they are *not increased* in number. On the contrary, they are, as a rule, somewhat diminished; ranging usually between 4,000 and 6,000 per c.mm. They may, however, at the beginning of the disease, be increased to 8,000 or even 12,000–13,000. A pronounced rise in the leukocytes sometimes marks the advent of complications. There is also a gradual decrease in the relative proportions of the neutrophile polynuclear cells and an increase in the lymphocytes.

As has been said, the blood contains the specific typhoid bacilli in most, if not in all, cases.

Urine.—The urine at first is concentrated, scanty and high colored. It frequently shows a trace of albumin and a few casts. It may contain blood, many casts and much albumin, from a complicating nephritis, or pus from a pyelitis or cystitis. Typhoid bacilli are found in it in about one-fourth of the cases. During the second and third weeks it usually, but not always, gives a positive response to Ehrlich's diazo-reaction (see Diagnosis).

COMPLICATIONS.—*Digestive Tract.*—The two most important complications are those of intestinal hemorrhage and perforation.

H e m o r r h a g e.—True intestinal hemorrhage occurs in from four to six per cent. of all cases. It is seen usually in the latter part of the second and in the third week, *i.e.*, at the time of the separation of the sloughs and the development of the ulcers. Occasionally slight bleeding from the congested mucous membrane occurs before the formation of the ulcers. The hemorrhage may be so slight as only to tinge a single stool or may be so persistent and copious as to prove fatal within a few hours. The symptoms of severe hemorrhage are a sudden marked fall in temperature, rapid, small pulse, pallor, restlessness, thirst, even syncope. The blood may not be passed until some hours after the symptoms have appeared, but usually appears promptly in the stools. Hemorrhage is always a serious complication; many of the cases are fatal, and the danger of further bleeding is always present for many days. Bleed-

ing from hemorrhoids or fissures in the rectum must not be confused with true intestinal hemorrhage.

P e r f o r a t i o n of the intestine is the gravest of the complications of typhoid fever. It occurs in from two to five per cent. of all cases and is almost always fatal from the septic peritonitis which the escape of intestinal contents excites. It occurs directly from extension of the typhoid ulcers through the gut and is seen usually during the third and fourth weeks of the disease. It may, however, happen in the second week or during convalescence. The site of the perforation is usually the lowermost part of the ileum, but it occasionally occurs in the colon or in the vermiform appendix. It is seen in both severe and mild cases. Tympanites, vomiting and coughing are regarded as contributory factors.

The *symptoms of perforation* are of the utmost importance, since immediate operation furnishes the only hope of recovery. They usually, but not always, begin abruptly and characteristically with sudden, severe abdominal pain, vomiting, tenderness and muscular rigidity—all symptoms of the beginning peritonitis. There is sometimes a temporary drop in temperature and a rapid rise in pulse-rate. As the peritonitis progresses the fever rises, the abdomen becomes greatly distended, the eyes sunken, the features pinched and cold, the breathing rapid and shallow and the general condition rapidly worse. The symptoms of perforation may be indistinct or lacking in cases in which the typhoid symptoms themselves are very severe. A rapid rise in the number of leukocytes and the replacement of the normal liver dulness by tympany are sometimes valuable aids to the diagnosis of perforation, but in most instances the leukocyte change is too slow to be of any value, and the liver dulness disappears only in the late stages.

To be of any avail the diagnosis of perforation must be made at the outset, chiefly on the basis of sudden, sharp pain in the abdomen, and abdominal rigidity, especially on the right side, and, in the clear-minded patient, pain.

Rarely septic peritonitis has developed without there having been actual perforation.

Suppurative parotitis and acute gastritis are infrequent complications. Acute glossitis, ulceration and subsequent stricture of the esophagus and abscess of the liver are among the rarities. An important group of complications are those affecting the gall-bladder and ducts. Acute cholecystitis and cholangitis due to infection by the typhoid bacilli occur, and gall-stones are a not infrequent sequel to the disease. Membranous colitis is a rare complication.

Circulatory System.—Myocarditis, pericarditis and endocarditis are rare, as is also thrombosis of the larger arteries. Acute dilatation of the heart may occur in convalescence. *Thrombosis of the large veins*, especially of the femoral, is much more common and is due to a localized phlebitis. This complication is usually not serious, but occasionally fragments of such a thrombus have been carried to the lungs and have caused fatal pulmonary embolism.

Respiratory System.—A severe *acute laryngitis* is sometimes met with. This may go on to ulceration and to necrosis of the cartilages. *Acute bronchitis* is very common and occasionally is so severe as to be a serious complication.

Lobar pneumonia occurs rarely as an initial symptom and not infrequently as a complication of the later weeks of the disease. *Hypostatic congestion* of the posterior portion of the lungs is not uncommon as a result of the long-continued dorsal decubitus and the enfeeblement of the circulation.

Pleurisy, both serous and purulent, is occasionally seen.

Urinary System.—*Acute nephritis* is an occasional complication and gives its characteristic symptoms. It occurs both early and late in the disease and may be of all grades of severity. Pyelitis and cystitis, due to the bacillus typhosus, are sometimes encountered.

Nervous System.—Neuritis occurs, usually as a late complication, in both a localized form and as a multiple neuritis affecting several extremities, but chiefly the legs. It is both sensory and motor. The commonest type of localized neuritis is that which causes the exquisitely tender toes and feet not infrequently seen in the latter half of the disease. Post-typhoid insanity is an infrequent sequel.

Skin.—Boils and subcutaneous abscesses are very common. Sometimes profuse sweating is met with.

Bed sores develop over the bony prominences—sacrum, elbows, heels, etc.—where emaciation is marked, and especially where the skin is irritated by the involuntary discharge of urine and feces. The hair is apt to fall out during convalescence, but always returns.

Miscellaneous Complications.—Otitis media is fairly common. Pyemia, arthritis, orchitis and intramuscular hemorrhage are all occasionally met with.

Among the commoner *sequellæ* are to be mentioned periostitis and necrosis of the long bones. They develop sometimes months after recovery, are chronic in course and frequently contain typhoid bacilli.

"Typhoid spine" is an obscure spinal affection occurring during convalescence and marked by severe pain in the back and other symptoms of vertebral disease, possibly due to periostitis.

The Association of Typhoid Fever and Malaria.—Typhoid fever, developing in one who previously has had malaria and in whom some organisms still remain quiescent, will sometimes cause the outbreak of fresh malarial paroxysms with the appearance of parasites in the peripheral circulation. Such cases are, however, very rare. The term "typhoid-malaria," so often loosely used, is confusing and misleading and should be avoided.

Diagnosis.—The diagnosis in typical cases is easy and can usually be made definitely by the beginning of the second week. The continued fever, the relatively slow pulse, the rose spots, the enlarged spleen, together with the absence of physical signs suggestive of other trouble, are usually conclusive. In the many variations from the typical course,

however, the difficulties of diagnosis may be very great and a correct judgment can be reached only by a careful weighing of all the symptoms and physical signs. No one symptom can be trusted entirely.

LABORATORY AIDS TO DIAGNOSIS.—The most useful of these is the so-called *Widal reaction*. This is based on the fact that the blood serum of typhoid patients, even if highly diluted, when brought in contact with actively motile typhoid bacilli causes prompt cessation of the motion of the bacilli and their collection into small groups or clumps. The technical details and the limitations of the test cannot here be given. A positive reaction is obtained in more than 95 per cent. of all cases of typhoid and it is very rarely found in other diseases, so that its presence is of very great diagnostic value. Unfortunately it is usually not present until the 8th or 10th day and it may not appear until the latter part of the disease.

Cultures.—Typhoid bacilli appear in the blood and in the feces usually by the end of the first week and sometimes earlier. Their separation from other bacteria in the stools is troublesome, but their detection in the blood is sufficiently simple to make it a practical and very valuable means of diagnosis in well-appointed hospitals. From 8 to 10 c.c. of blood are drawn, under due aseptic precautions, from the median basilic vein and inoculated usually in two bouillon flasks. The bacilli, if present, appear in from 24 to 48 hours.

Ehrlich's diazo-reaction in the urine is present in most cases of typhoid, but it is occasionally met with also in other febrile conditions, such as acute tuberculosis. Its presence, therefore, while strongly suggestive, is not conclusive of typhoid. The absence of an increase in the leukocytes in typhoid is often of considerable help in differential diagnosis. The disease is apt to go unrecognized in those cases in which the initial symptoms are abnormally severe in certain of the viscera. Such cases are often mistaken at first for severe bronchitis, lobar pneumonia, nephritis or meningitis.

Typhoid fever and malarial fever should not often be confused. Only the remittent or estivo-autumnal type of the latter bears a resemblance to typhoid. In doubtful cases examination of the blood for malarial parasites and for the Widal reaction should clear up the diagnosis.

Other diseases that may be confused with typhoid are, typhus, septicemia, acute miliary tuberculosis, tuberculous peritonitis, malignant endocarditis, influenza, salpingitis and, rarely, appendicitis.

Prognosis.—The mortality varies greatly in different years and in different epidemics. In general it ranges between 5 and 20 per cent. In recent years it has been much reduced by the improvement in nursing and by the introduction of the cold bath treatment. The death rate is lowest in children and highest in those of advanced age. It is much lower in relapses than in the primary attacks.

The cause of death in almost half of the fatal cases is the severity of the systemic poisoning, as shown by hyperpyrexia, severe nervous symptoms and heart weakness. Next in order of importance are intestinal

hemorrhage, perforation and peritonitis, pneumonia and acute nephritis.

Treatment.—Prophylaxis.—Typhoid fever is a preventable disease. The fact that the exciting germs leave the body only through the stools, urine and vomited matter and enter the body only through the mouth and stomach renders the task of prevention a relatively simple one. The responsibility for proper and thorough disinfection in every case rests with the physician. The solutions most useful for the disinfection of the discharges are (a) carbolic acid (1 to 20), (b) corrosive sublimate (1 to 500) and (c) formalin (5 per cent.). The feces and urine should be received into vessels containing an abundance of the disinfectant solution, should be thoroughly mixed with it and should be allowed to stand one hour before being disposed of. Under no circumstances should the discharge be thrown into privies, cesspools or upon the ground without such disinfection. All bedding and linen should be soaked in carbolic acid (1–20) for several hours and subsequently thoroughly boiled. The buttocks and perineum should be carefully cleaned after each defecation with corrosive sublimate solution (1–2000). The hands of the nurses, thermometers, bed-pans and everything which may come in contact with the discharges should be as carefully disinfected. Disinfection should begin as soon as typhoid fever is suspected and should continue well into convalescence.

Since infection occurs in the great majority of cases by means of the drinking water the simple precaution of boiling this thoroughly removes all chance of infection through it. If there is reason to fear that the milk may be contaminated it should not be used uncooked. The same may be said of oysters. Ice from polluted streams should not be used. Where open privies and cesspools abound flies may be important carriers of the infection and the greatest care should be exercised to prevent them from reaching the articles of food.

Preventive inoculations have very recently been used in the British Army and promise to be of real value. The vaccine consists of a broth culture of the bacillus typhosus in which the organisms have been killed by heat.

General Management.—In no disease is careful attention to the details of management and nursing so richly rewarded as in typhoid fever. The patient should, even in mild cases, be put to bed at once and kept there throughout the disease; the use of the bed-pan and urinal being insisted upon. Whenever possible a single bed with firm, even surface should be used, as it greatly increases the patient's comfort and facilitates his handling. The room should be quiet, cool and well ventilated. The keeping of the bed linen scrupulously clean, free from wrinkles and dry, especially when there is incontinence of urine or feces, will do much to prevent bed sores. To the same end the patient's back should be kept clean and dry, rubbed frequently with alcohol and protected by dusting powder or a bland ointment. The mouth should be carefully cleansed after each feeding. At the outset the bowels should be freely moved by calomel or castor oil. After the first week

it is wiser to keep them regular by enemata or suppositories. The patient is not permitted to leave the bed, except perhaps in very mild cases, until the temperature has been normal for at least a week. Water to drink should be given at frequent, regular intervals and in large quantities.

DIET.—Food should, in general, be fluid and easily digestible and should be given at two-hour intervals, except during the night. In most cases milk is the article chiefly to be relied upon. From three pints to two quarts per day are usually required. It is frequently better digested if diluted with vichy, lime water or plain water, or if partly peptonized. Strong meat broths (chicken, mutton, beef), buttermilk, kumyss, egg-water, beef juice, thin strained gruels, coffee, and ice-cream may be given to vary the feeding. Gelatinous foods, such as calf's-foot jelly, have both a theoretical and practical value. Rarely the artificial milk foods and ''peptonoids'' may be useful. With the beginning of convalescence the appetite returns and the hunger is often such as to cause real suffering. The articles mentioned can be given in larger quantities and in greater variety, and to them may be gradually added soft boiled eggs, milk toast, junket, custards, scraped meat, bread and butter, toast, as the returning appetite and increasing power of digestion call for them. Fever is no contra-indication to the giving of solid food, provided it be given in small quantities and the patient is able to masticate it thoroughly. (See Appendix, p. 592.)

MEDICINAL TREATMENT.—Attempts to find a specific treatment have not been successful. No drug is known which has a favorable influence upon the typhoid infection itself. Efforts to destroy the bacilli in the intestine by the use of salol, β-naphthol, carbolic acid, etc., have not succeeded, and it is quite hopeless to attempt by such means to reach the germs which swarm in the intestinal lesions, mesenteric glands, spleen and blood. Such drugs may sometimes perhaps be of use, as anti-fermentatives, in diminishing meteorism, but even here their usefulness is questionable. If intestinal antiseptics are to be used salol (gr. v every 3 hours) seems to be the best. The use of antipyretics, stimulants, etc., will be spoken of under treatment of the individual symptoms.

HYDROTHERAPY.—The treatment of typhoid by cold baths, under the leadership of Brandt of Germany, has in recent years done much to reduce the mortality and to rob the disease of some of its distressing symptoms. Such treatment is in no sense specific. It is doubtful if the disease is materially shortened by it. Nevertheless its favorable influence upon certain of the symptoms is so evident that it is at present in very general use both in this country and in Europe. The reduction of the temperature is by no means the chief effect of such baths. Their influence in strengthening the heart action and in improving the serious nervous symptoms is even more important.

Hydrotherapy may be practiced by cold sponging, wet packs, bed baths or tub baths. The first three methods are to be recommended only in case the tub baths, for any reason, are impracticable.

Tub Baths.—A portable tub, large enough for the patient to lie at full length, is placed near the bed. The patient is lifted to and from the bath, which is given whenever the temperature (taken every three hours) rises above 102.5° or 103° F. As to the temperature of the bath, practice varies greatly, but the very cold baths (60°–65°) are much less used than formerly. It is rarely desirable to have the temperature lower than 72° and most of the good effects can be obtained by baths of 75° or 80°. The patient's comfort is greatly increased by beginning the bath at 90° and gradually cooling the water by adding ice. The bath should last for 15 minutes and the patient should be vigorously rubbed by two attendants throughout that time. During this time also it is well to keep the head wet with cold water. It is often desirable to precede the bath by the administration of half an ounce of whiskey or other stimulant. At the end of the bath the patient is wrapped in a dry sheet, covered with a light blanket, given a hot drink and encouraged to sleep. If the baths are being well borne the blueness and shivering soon disappear and the patient usually falls into a quiet, restful sleep. Intestinal hemorrhage, perforation and severe lobar pneumonia are the only positive contra-indications to such baths. Other pulmonary complications, such as bronchitis, are often greatly benefited.

Where tub bathing is not practicable sponge bathing or cold packs may be resorted to. They, however, are much less efficient and have often proved more unpleasant than the baths above described.

TREATMENT OF SPECIAL SYMPTOMS.—*Fever.*—The most effective and at the same time the safest way of reducing the temperature is by use of the cool baths already described. The fall is usually from one to three degrees and it is often several hours before the fever reaches its former height. In private practice it is often desirable to prescribe some mild febrifuge such as the following: ℞ Potassii citratis, ℥vi; spt. ætheris nitrosi, ℥iss; aqua, q. s. ad ℥vi. S. One dessertspoonful in cold water every four hours. The use of antipyretic drugs for this purpose has, happily, to a considerable extent been abandoned. The continued use of such drugs as antipyrine and phenacetin in doses sufficient to materially affect the fever is often accompanied by such cardiac depression, pallor, cyanosis, etc., as entirely to nullify the good effects of the reduction of temperature, and even threaten life. If any antipyretic drug is to be used, aspirin or quinine is the least objectionable.

Diarrhea requires treatment only if the number of movements daily exceeds three or four. It may often be corrected by some change in the diet. If drugs are required bismuth subnitrate (gr. xx–xxx) or pills of acetate of lead (gr. ij) and opium (gr. ss) are usually efficacious. When there is tenesmus a starch enema will often give relief.

Tympanites.—Efforts should be made to prevent tympanites by careful attention to diet. When present nothing, in my experience, is so likely to give relief as very hot turpentine stupes to the abdomen applied for 15 minutes every hour or two. The frequent passage of a long rectal tube is sometimes of great service. The good effects of intes-

tinal anti-fermentatives (turpentine, salol, charcoal, etc.) may occasionally be seen, but they are usually disappointing. In severe cases cessation of feeding for 12 to 24 hours is helpful.

Intestinal Hemorrhage.—Treatment is limited chiefly to careful management of the patient. He should have absolute quiet and freedom from all movement. Food should be stopped altogether for 12 hours or else given in very small quantities. Pills of opium, or morphine subcutaneously, should be administered to check intestinal peristalsis. Ice to the abdomen is of doubtful value. There is little evidence that the internal use of the various astringents and styptics ever does good. Acetate of lead and suprarenal extract are commonly recommended, but are useless. Bismuth in very large doses may, perhaps, be of service. Subcutaneous injections of gelatin in the effort to promote coagulation have been tried, but have been given up. If the bleeding has been very profuse, heat to the body surface, elevation of the foot of the bed, subcutaneous infusion of normal salt solution and vigorous stimulation may be required, but these last two measures should not be used unless imperatively needed, for fear of exciting further hemorrhage.

Perforation.—Whenever intestinal perforation is suspected bathing should be stopped at once, all food should be temporarily withheld and absolute quiet maintained. Early operation offers the only hope of relief. About one-fourth of the cases recently operated upon have been saved. Since the chances of success are diminished by each hour of delay, the importance of prompt diagnosis is manifest. If necessary the operation may be done under cocaine.

Circulatory Failure.—Experimental study has shown that in animals rapidity and weakness of the pulse and evidences of circulatory failure developed in the course of bacterial infections result not from weakness of the heart but from disturbances in the vasomotor system produced by the toxins of the disease. Also against such circulatory failure the traditional cardiac stimulants, alcohol, digitalis, and strychnine, have no influence, but camphor, caffeine, applications of cold to the splanchnic region, or infusions of normal salt solution have some effect.

These teachings have special application to the circulatory failure of typhoid fever. It is undoubtedly true that in most cases of such failure the heart can be heard and often felt beating regularly and energetically, and yet the pulse is rapid, feeble, perhaps uncountable, the body cyanotic, and the patient evidently suffering from circulatory failure. Under such circumstances, caffeine, camphor, and the other measures mentioned above are indicated. Camphor should be given hypodermatically dissolved in olive oil, not in ether, the latter solution being much too irritant. One or two grains of camphor may be given every 2 hours.

Caffeine-sodium salicylate or caffeine-sodium benzoate may be used hypodermatically in doses of 2 grains or more every 2 or 3 hours.

Infusions of normal salt solution 300 c.c.–500 c.c., or even 1000 c.c.,

may be given and repeated according to indications. An ice-cap or ice-coil should be kept applied upon the epigastrium.

If the heart itself seems to be at fault then digitalis, strychnine, or alcohol may be given. The use of alcohol in infectious fevers is the source of much discussion. All the experimental evidence is against its use; yet most experienced clinicians still give it, sometimes in large quantities. One-half to one ounce of whiskey every three or four hours is the ordinary dose.

Nervous Symptoms.—All the severe symptoms are best relieved by cold baths. The early headache and sleeplessness may require phenacetin (gr. v–x), Dover's powder (gr. v–x) or trional (gr. x–xx).

Recrudescences.—The sudden rises in temperature during convalescence are most commonly due to constipation or to dietetic errors. Fluid diet should be at once resumed and, if there is no contra-indication, a laxative, preferably castor oil, given. One is often amazed at the amount of accumulated feces that such a laxative will bring away, even when the diet has been entirely fluid.

TYPHUS FEVER

(Spotted Fever.   Jail Fever.   Typhus Exanthematicus)

Definition.—This is an acute, highly contagious, infectious disease marked by an abrupt onset, a febrile course of about two weeks, a characteristic rash and severe nervous symptoms.

Etiology.—Predisposing Causes.—Typhus fever is at the present day a comparatively rare disease. It occurs endemically in England, Ireland, Russia, Hungary and in various parts of Asia and it appears in other countries sporadically and in epidemics. A few epidemics have occurred in the large seaboard cities of this country. Overcrowding, poor food and filth are potent predisposing factors. It is therefore most frequently seen in camps and barracks, jails and in the crowded and unsanitary portions of large cities. It follows in the path of war and famine. Most epidemics occur in the colder months of the year. The disease attacks all ages and is seen in males more commonly than in females.

The direct, exciting cause is unknown, but it is doubtless a microorganism of some sort. The disease is one of the most contagious known, the contagion being transmitted, as in other such diseases, by the expired air, by exhalations from the skin, by clothing, bedding, etc. Its contagiousness is greatest in close, crowded rooms and least in the open air and in large, well-ventilated wards. It is probably never transmitted through food and drink.

The infectious agent has been proven to exist in the blood of the patient, the disease having been transmitted to monkeys in series by inoculations of infected blood. The pediculus vestimentorum is regarded as the probable agent by which the disease is conveyed from man to man. Predjetschensky has obtained from the blood drawn on

the 6th to the 9th day of the fever a short, thick bacillus, which he regards as the causative organism. Sufficient time has not yet elapsed to allow proper testing of these plausible propositions.

IMMUNITY for life is usually provided by one attack.

Morbid Anatomy.—Aside from the petechial rash which persists after death, the lesions found at autopsy are in no wise characteristic. They are those of any severe infection—dark fluid blood, parenchymatous degeneration of heart, liver, kidneys and voluntary muscles, hypostatic congestion of lungs, etc.

Symptoms.—THE PERIOD OF INCUBATION is quite regularly about twelve days. It may, however, be as short as one week or as long as fifteen days. Prodromal symptoms are usually lacking.

ONSET.—The disease begins abruptly with one or more chills, vomiting, severe headache, backache and pain in the legs. Prostration is marked from the outset. The eyes are red and watery and the face flushed and swollen. The tongue is dry almost from the beginning and is heavily coated.

FEVER.—The temperature rises steadily during the first three or four days to 104° or 105° F., or even higher. It then maintains this height with only slight daily fluctuations until the latter part of the second week (12th to 15th day), when it falls rapidly to normal. This period of defervescence usually occupies two or three days. The fall may, however, be by crisis. Just before defervescence the temperature may be especially high (106° to 107° F.) or there may be a temporary fall or pseudo-crisis.

ERUPTION.—The rash is one of the characteristic and constant features of the disease. It appears quite regularly on the 4th or 5th day. It may be accompanied or preceded by a peculiar dusky mottling, or a measles-like blotching of the skin, which lasts only a day or two. The true rash consists of a single crop of pale, pinkish macules of pinhead size or somewhat larger, which at first disappear on pressure. These appear over abdomen and chest and rapidly extend to back and limbs; the palms of the hands, soles of the feet and face alone remaining free. Within two or three days blood escapes into most of these hyperemic areas, they become a deeper red, and no longer disappear on pressure.

The *petechial* character of the rash constitutes its chief diagnostic feature. The spots which do not become petechial disappear in three or four days, but the petechiæ remain for a week or ten days, gradually fading to a dirty, brownish stain. Exceptionally the rash is petechial from the outset and may be accompanied by subcutaneous hemorrhages of some size. The eruption is usually more abundant than that of typhoid, which it at first so closely resembles. Moderate enlargement of the spleen is usual and occurs as early as the second or third day.

The PULSE is much quickened almost from the beginning, its rate being usually between 110 and 120 per minute. At first the pulse is full and tense, but in the second week it grows steadily smaller and

weaker and may become almost imperceptible. Dicrotism is much less common than in typhoid. The bowels are usually constipated, but diarrhea is not uncommon during the second week.

The NERVOUS SYMPTOMS are among the most conspicuous of the disease. The severe headache of the onset soon gives place to mental apathy and this, in many cases, to violent delirium. In the second week this delirium merges into deep stupor, with vacant, staring eyes, muttering speech, wide open mouth and trembling, dry tongue (coma-vigil). The patient is entirely oblivious to his surroundings. Urine and feces are passed involuntarily and there are involuntary twitchings of the muscles of the extremities (subsultus tendinum), and purposeless movements of the cyanotic, shrunken fingers. In the milder cases there may be only apathy and drowsiness with quiet, nocturnal delirium. The urine shows no characteristic change. A small quantity of albumin is not uncommon.

In contradistinction to typhoid a distinct increase in the leukocytes of the blood is usual, but leukopenia may be found.

Variations from the course described are of frequent occurrence. Mild cases of short duration are frequently seen, especially toward the end of an epidemic. Malignant cases, fatal within a few days, are met with. Rarely, too, cases of three weeks' duration are encountered.

CONVALESCENCE.—With the critical fall of temperature there is a correspondingly rapid improvement in the other symptoms. The skin is bathed with profuse perspiration. The stupor and delirium give place to a refreshing sleep, the pulse becomes slower and stronger. Relapses are extremely rare and later complications not very common, so that the convalescent patient usually progresses steadily and fairly rapidly to recovery.

Complications and Sequelæ.—Pulmonary complications are the most important and include severe bronchitis, bronchopneumonia, hypostatic congestion and gangrene; otitis media, laryngitis, gangrene of the extremities, venous thrombosis, nephritis, peripheral neuritis and hemiplegia, and subcutaneous abscesses also occur.

Diagnosis.—The disease during its early stages is often extremely difficult to diagnosticate. Small-pox, scarlet fever, measles, meningitis and typhoid fever all may be confused with it. A careful study of all the symptoms, and especially of the fever and the nature of the eruption, is needed to clear away the doubt. From typhoid fever the disease is differentiated by its more rapid and violent onset, the swollen face and suffused eyes, the increase in leukocytes if present, the absence of the Widal reaction, negative blood cultures, and especially by time of appearance, distribution and character of the eruption. This appears earlier, is more abundant on the arms and legs, does not come out in successive crops, is less distinctly elevated and, in the course of two or three days, becomes petechial, at least in part, and then no longer disappears upon pressure. In fatal cases the absence of the post-mortem lesions characteristic of typhoid fever is most important.

Prognosis.—The mortality varies greatly in the different epidemics. In general it may be said to amount to 15 or 20 per cent. In single epidemics it may reach 50 per cent. The death rate is lowest in children and highest among the debilitated, the alcoholic and aged.

Treatment.—The highly contagious nature of the disease demands the most careful isolation and disinfection. Free ventilation is known to diminish greatly the chance of contagion for those in attendance as well as to be beneficial to the patient himself. No specific treatment is known. The use of drugs should be limited to the treatment of such special symptoms as heart weakness, cough, etc. For the fever and the severe nervous symptoms hydrotherapy (preferably by tub baths) is the most rational treatment. This, as well as the dietetic treatment, should be carried out along the lines suggested for typhoid fever.

RELAPSING FEVER

(Recurring Fever. Famine Fever. Seven-day Fever)

Definition.—An acute infectious disease caused by a specific microorganism, the spirillum of Obermeier; and marked by two or more severe febrile attacks, each of several days' duration, which are separated by afebrile intervals of similar length.

Etiology.—The disease at the present day is rare. Epidemics appear from time to time in various parts of Europe and in the British Isles. It is said to be common in India. A few epidemics have occurred in New York and Philadelphia—the last one in 1869.

In its PREDISPOSING CAUSES it is closely allied to typhus fever in that filth, overcrowding and famine are the conditions which chiefly favor its development. Age, sex, and season seem to have little influence.

The EXCITING CAUSE is a blood parasite, a spirillum or spirochete, which was first seen by Obermeier in 1873. It is a slender, actively motile, spiral filament whose length is several times the diameter of a red cell. These germs are constantly present in the blood plasma during the febrile paroxysm, but disappear just before the crisis and are not to be seen during the intervals. They have not been found in the body secretions or elsewhere than in the blood. Little is known of their life history. The disease has been transmitted to man and monkeys by the inoculation of infected blood. It is believed to be contagious by direct transmission and by means of fomites, but in the light of our present knowledge of the mode of transmission of malaria and yellow fever it seems possible that direct inoculation into the blood, by insects or other means, may be found to be necessary.

Morbid Anatomy.—No characteristic lesions are found after death. There are parenchymatous degeneration of the viscera, swelling of the spleen and sometimes jaundice.

Symptoms.—The INCUBATION PERIOD commonly lasts from five to seven days. It may, however, be considerably shorter or longer.

The ONSET is sudden, with a distinct rigor and an immediate rise of

temperature. There are also pains in the head, back and limbs; vomiting and prostration. Within a few hours the fever rises to 104° or 105° F., and then remains constantly high (104° to 107° F.) for several days. Sweating, cough, delirium and jaundice may be present. The pulse is rapid and full; the spleen becomes enlarged early and the blood shows an increase in leukocytes. Herpes labialis is sometimes seen. In severe cases cutaneous petechiæ and ecchymoses may occur. (See Fig. 64.)

On the 6th or 7th day, usually, the paroxysm ends by CRISIS. With the sudden fall of temperature to normal or below there is profuse sweating and sometimes diarrhea. In debilitated subjects the collapse may be profound. The temperature remains normal in most cases for about a week and there is a corresponding remission in all the other symptoms; then the patient again has a chill (or several chills) and a rise of temperature, and the first attack is repeated except that the duration is apt to be somewhat shortened. After a second crisis and afebrile period a third, and even a fourth and fifth paroxysm may occur.
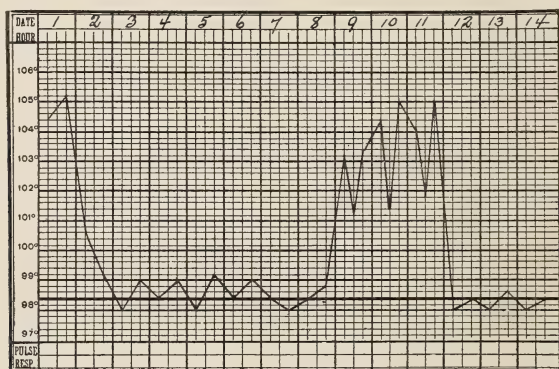
Fig. 64.—Temperature of relapsing fever, showing the conclusion of one paroxysm. The afebrile period, and a new paroxysm.

Variations from the above typical course are frequent as regards the duration of both the febrile attacks and the free intervals. In some mild cases no relapse occurs. In severe cases jaundice may be a conspicuous feature.

Complications.—There may be pneumonia, nephritis, neuritis, ophthalmia, hemorrhage of stomach, intestines or kidney, or rupture of the enlarged spleen.

The PROGNOSIS is usually good. The death rate is low (4 per cent.), but varies much in the different epidemics.

Diagnosis.—In its onset relapsing fever cannot be distinguished from a number of other infectious diseases. Its later course, however, is so characteristic that it can hardly be misinterpreted and a positive and easy means of diagnosis is at hand in the examination of the blood.

The parasites disappear from the blood in the afebrile stage, but Lowenthal has shown that if a drop of blood from a patient in this

stage be mixed with a drop from another patient in the febrile stage and therefore containing the spirochetæ, the preparation sealed and placed in a thermostat for one-half hour, the organisms lose their motion. In this manner mild or abortive types of the fever may also be recognized.

Treatment.—Drugs seem to have no effect upon the course of the disease. The cases must be treated symptomatically along the lines suggested under typhoid fever. Isolation and disinfection should be rigidly enforced. Injections of arsenobenzol, 0.3–0.5 gm., as for syphilis (see p. 450), are said to be effective.

INFLUENZA
(La Grippe)

Definition.—An acute febrile epidemic disease, due to a specific micro-organism, and characterized usually by catarrhal symptoms, by great prostration, by inflammation of the various mucous membranes, especially of those of the respiratory tract.

Etiology.—The disease has for centuries occurred in vast epidemics which, beginning usually in Russia, have spread rapidly over the greater part of the world. For several years after such an epidemic many sporadic cases are seen, especially in the colder months of the year. The disease attacks all ages and both sexes indiscriminately. The direct exciting cause is the *bacillus influenzæ*, a very small, non-motile organism discovered by Pfeiffer in 1892, found abundantly in the secretions from the inflamed membrane.

Morbid Anatomy.—Aside from a catarrhal inflammation of the affected mucous membranes no lesions are found *post mortem* except those of the various complications.

Symptoms.—The PERIOD OF INCUBATION is usually from two to four days. The ONSET is quite abrupt, with a sudden rise of temperature to 103° or 104° F., chilliness, headache, severe pain in the back and limbs and, usually, great prostration. (See Fig. 65.) According to the predominance of special symptoms the cases are divided into the CATARRHAL TYPE, with symptoms of coryza, laryngitis or bronchitis; the NERVOUS TYPE, in which the chief symptoms are severe headache, insomnia, neuralgia, and pain in the joints; the GASTRO-INTESTINAL TYPE, with persistent nausea and vomiting or severe diarrhea, and the FEBRILE TYPE, in which high fever is almost the only symptom.

Course.—In uncomplicated cases most of the symptoms subside in from three to seven days, although the prostration which is often so prominent a feature may persist much longer.

Complications.—The disease is marked by a tendency to the development of a great variety of serious complications, of which pneumonia is much the most important. Influenzal pneumonia, although similar to lobar pneumonia in its histological features, differs from the latter clinically in its association with extensive bronchitis, its tendency to develop irregular areas of consolidation in both lungs, its frequent defervescence by lysis, and its higher mortality. Severe bronchitis fre-

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quently occurs without evident pneumonia. Other occasional complica-
tions are otitis media, pleurisy, endocarditis, pericarditis, cardiac irreg-
ularity of obscure nature, venous thrombosis, nephritis, meningitis,
neuritis, melancholia, etc., etc.

Diagnosis.—Great caution should be exercised in the diagnosis when
no epidemic prevails. The sudden violent onset, severe pains and pros-
tration are the features most to be relied upon. The term ''grippe''
is loosely and improperly applied to many cases of simple coryza, ton-
sillitis, bronchitis, etc., which bear no relation to true influenza.

The only satisfactory basis for the diagnosis of influenza is the
demonstration of the Pfeiffer bacillus in large numbers in the sputum,
or other secretion, such as the cerebrospinal fluid. On this basis influ-
enza is now a rare infection. The great majority of cases to which the
name is ordinarily applied are infections by other organisms, such as
the staphylococci, streptococci, the micrococcus catarrhalis, etc.
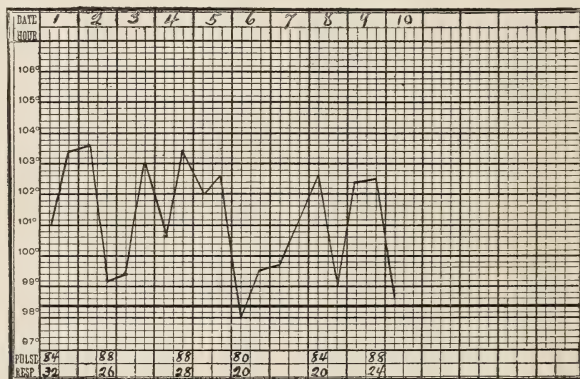
Fig. 65.—The irregular fever of influenza.

Prognosis.—The prognosis of true influenza varies with the age
and general condition. During epidemics the disease proves very fatal,
especially through the complicating pneumonia. The weak and debili-
tated, especially elderly people, frequently succumb. In the young and
vigorous influenza is rarely fatal, but protracted anemia and debility
often follow.

Treatment.—The patient should immediately be put to bed and
upon a fluid diet, and a brisk purge given. The fever and the pains
are usually best combated by the combination of an antipyretic and
a salicylic compound, e.g., phenacetin and salol, or aspirin 5 grains
every three or four hours. If cardiac depression be marked, stimula-
tion by strychnine, caffeine or alcohol may be needed. When gastric
symptoms are prominent, it is often necessary to withhold all medication
for a time and to give fluid food only in minute and frequently repeated
doses. Removal to a milder climate and rest are often necessary to
convalescence.

DENGUE

(Break-Bone Fever)

An epidemic, infectious disease of warm climates marked by one or more paroxysms of fever, intense pains in the bones and joints and a skin eruption.

Etiology.—Epidemics of dengue have been seen in various tropical and sub-tropical countries, and a number have occurred in the Gulf States of this country. The disease resembles influenza in its rapid spread and in the fact that it attacks a large proportion of the population of all ages. Evidence is accumulating to show that the mosquito is the chief means of transmission, and a parasite similar to the plasmodium of malaria has recently been found in the blood cells. The disease usually appears during the warm months of the year.

Nothing is known of its morbid anatomy, since it is rarely fatal.

Symptoms.—The INCUBATION PERIOD is from two to five days. The onset is very sudden, with intense pains in the joints and bones, headache, and a rapid rise of temperature to 104° or 105° F. or even higher. The pulse is proportionately rapid, the tongue coated, the face flushed and swollen, the urine scanty and the bowels constipated. A transient erythematous rash is often seen at the beginning of the disease. The pains attack many of the joints, which are usually also tender and swollen. At the end of three or four days there is a marked remission for two or three days of the temperature and the agonizing pains, after which the fever and pains return for two or three days and there is usually also an eruption, which may be measly, scarlatiniform or urticarial in character. At the same time there is often enlargement of the lymph glands. Hemorrhages from the mucous membranes occur rarely. The disease usually lasts from seven to ten days, although prostration, pains and stiffness may persist for some time longer. The PROGNOSIS is perfectly good, fatal cases being almost unknown. Complications are rare. One attack does not afford complete immunity.

Diagnosis.—The epidemic character, the prominence of the joint symptoms and the late eruption are the features of special diagnostic value. The disease may be confused with influenza, acute articular rheumatism, epidemic meningitis or yellow fever.

Treatment.—The diet should be fluid. Morphine hypodermically is often needed to relieve the excruciating pains. Antipyrine or phenacetin may suffice in the less severe cases. Very high temperature or delirium demands cold sponging or packs. If prostration is marked stimulation by alcohol or strychnine may be needed.

YELLOW FEVER

Definition.—Yellow fever is an acute, non-contagious, infectious disease marked by a sharp febrile paroxysm, albuminuria, jaundice and a tendency to hemorrhage, especially into the stomach. It is transmitted by the bite of certain infected mosquitoes.

Etiology.—The disease is endemic only in a few well-defined tropical districts, which include certain of the sea-ports of the West Indies, of Central America and of tropical South America, and certain parts of the coast of West Africa. From these foci it occasionally spreads in large epidemics to temperate regions and especially to the southern portion of the United States. It is rarely seen at altitudes of above 1000 feet and epidemics are usually promptly checked by frost. Yellow fever attacks both sexes and all ages, but is usually seen in young adults. The negro race is relatively immune. Its transmission has been definitely proven to be by means of infected mosquitoes of the species *Stegomyia fasciata,* and not, as has always been supposed, by mere personal contact or by infected fomites (see Fig. 66).

The germ of yellow fever is unknown. However, the Yellow Fever Commission of the United States Marine Hospital Service has offered much evidence to show that it may be an animal parasite, closely related to the malarial plasmodium, which finds its intermediary host in the mosquito above mentioned.

Morbid Anatomy.—The post-mortem findings include deep jaundice of the skin and tissues generally, petechial and larger hemorrhages into the skin, muscles and serous and mucous membranes, and acute parenchymatous degeneration of the liver, kidneys, and heart. The liver especially shows marked changes. It is usually small and of a pale brownish color and reveals extensive fatty degeneration of its cells with areas of focal necrosis. The diagnosis of yellow fever cannot safely be made from the autopsy findings alone.

Symptoms.—The PERIOD OF INCUBATION lasts from one to five days. The symptoms are usually divided into three periods.

(1) STAGE OF INVASION, OR FEBRILE STAGE.—The onset is abrupt and is marked by a distinct chill. The fever rises rapidly to 103° or 104° F., the pulse is rapid and bounding, there are headache, pain in back and limbs, nausea, vomiting and a hot, dry skin. The face has a peculiar swollen, flushed look, the eyes are injected and the sclerotics, even on the first or second day, usually reveal the beginning jaundice. The tongue at first is coated and moist, but soon becomes dry and brown. Vomiting usually persists and may even in this stage become hemorrhagic ("black vomit"). The urine is scanty and within a day or two becomes albuminous. The fever continues high, the pulse grows weaker but at the same time *becomes slower*. Jaundice may be slight or well marked. Distinct enlargement of the spleen is uncommon. This stage lasts from one to four days and is followed by

(2) STAGE OF CALM.—The fever rapidly falls to about normal, the vomiting ceases, the jaundice lessens and the patient is much more comfortable. This may mark the beginning of convalescence, but in many cases, after some hours or a day or two, the symptoms all return.

(3) STAGE OF REACTION.—The fever again becomes high and all the constitutional symptoms return. Jaundice deepens, hemorrhages occur beneath the skin and from the various mucous membranes, especially

into the stomach, whence the blood is vomited in the form of brownish, grumous material which constitutes the dreaded ''black vomit.'' The patient becomes delirious, the urine is often altogether suppressed, the
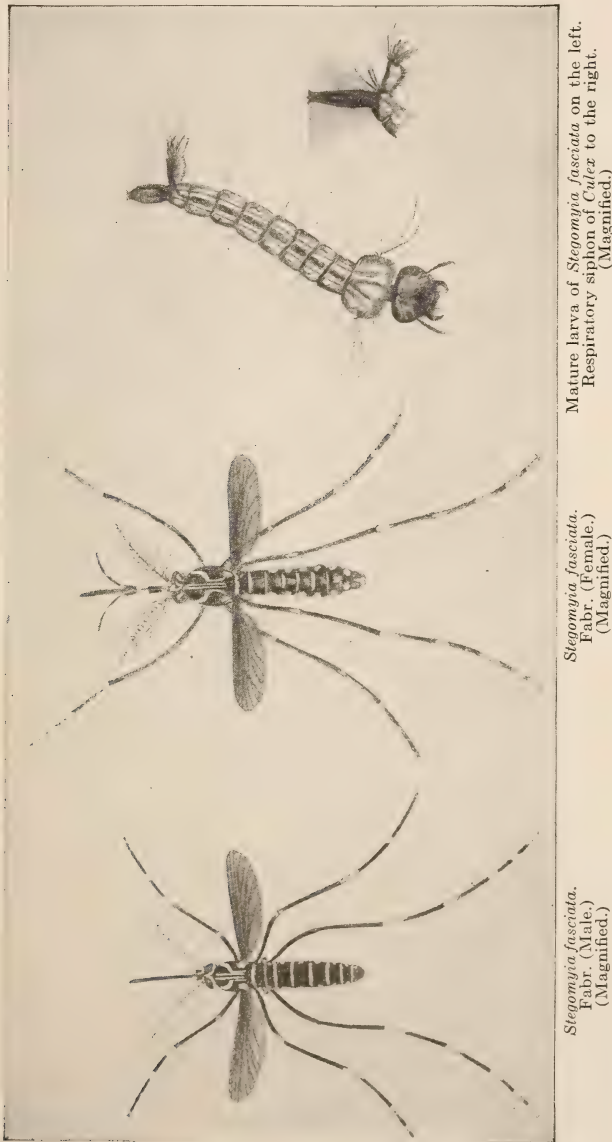
Mature larva of *Stegomyia fasciata* on the left. Respiratory siphon of *Culex* to the right. (Magnified.)

Stegomyia fasciata. Fabr. (Female.) (Magnified.)

Stegomyia fasciata. Fabr. (Male.) (Magnified.)

Fig. 66.—(International Clinics, Vol. I, Series 21.)

pulse is small and very feeble, but is often strikingly slow (60 to 80). Most of the cases which enter this stage die within one or two days. In the few that recover convalescence is apt to be slow and tedious. One attack usually confers immunity for life.

Diagnosis.—The features upon which the diagnosis is to be based are the violent onset, the characteristic facies, the early albuminuria and jaundice and the falling pulse rate with a high or rising temperature. The disease may readily be confused with the severer form of malarial fever, in which, however, the jaundice and albuminuria are less common and appear later, the spleen is much enlarged and the blood contains the malarial plasmodium. Dengue also may occasionally give a clinical picture closely resembling that of yellow fever.

Prognosis.—The mortality varies in different epidemics between 10 and 75 per cent. A very high temperature, suppression of urine and the black vomit are especially grave symptoms.

Prophylaxis.—The modern view that yellow fever is not contagious and is spread only by the bites of infected mosquitoes seems to show that the rigid quarantine hitherto in vogue is unnecessary. Rational prophylaxis lies in preventing those sick of the disease from being bitten by mosquitoes and in protecting healthy persons from the bites of any mosquitoes which may possibly be infected, and in destroying the mosquito so far as possible. The success of the United States authorities in checking the outbreak of 1905 in New Orleans by these measures and the prevention of outbreaks in Panama have thoroughly established the correctness of this theory.

Treatment.—This is purely symptomatic. An active purge should be given at the onset; the fever is to be combated by bathing or by cold packs and the heart supported by strychnine, digitalis and alcohol. Great care should be exercised in the feeding and in the administration of drugs to avoid irritation of the stomach. Only fluid foods such as broths or milk and vichy should be given and these only in small quantities. If vomiting is severe it may be necessary to withhold for two or three days all food and drink by mouth and to supply nourishment only by the rectum. For the tendency to hemorrhage little can be done. Calcium chloride by mouth or rectum and subcutaneous injections of gelatin may be tried.

Sternberg's plan of treatment has been generally approved. Sodium bicarbonate 2½ drams and bichloride of mercury gr. ⅓ are dissolved in 1 quart of water: of this solution an ounce and a half is given every hour.

THE PLAGUE

(Bubonic Plague. Pest)

Definition.—An epidemic infectious disease of great fatality, due to the invasion of a specific bacterium—*Bacillus pestis*—and marked clinically by severe constitutional symptoms and the development usually of suppurative buboes.

Etiology.—Bubonic plague, the "Black Death" of the Middle Ages, has been known as one of the great pestilences for many centuries. In London in 1665 it destroyed 70,000 lives, or more than one-seventh of the total population. The last great epidemic began in China in 1894

and for several years raged with great virulence throughout India. A number of cases appeared in San Francisco in 1890. In 1910 and 1911 the disease ravaged Manchuria and Northern China. It seems to be endemic in certain parts of Asia and in Uganda. Age, sex, and occupation have little influence upon susceptibility. It prevails chiefly in the hot months. Filth and overcrowding are the factors chiefly influential in determining its spread. It is therefore confined almost exclusively to the native population. Doctors, nurses and those living under proper hygienic conditions are rarely attacked. Infection probably occurs through both the digestive and the respiratory tracts and also through abrasions of the skin. Rats and mice are very susceptible to infection and are believed to play an important rôle in the spread of the disease. There is now much evidence that the bites of the rat-flea may be the chief means of infection. In Manchuria the disease was believed to be spread by the infected skins of the marmot. The ground-squirrels of California have been shown to be widely infected. The infection is probably conveyed through foods (such as rice) and water.

Bacillus pestis, discovered by Kitasato and Yersin in 1894, is a very short, thick rod with rounded ends, and is found abundantly in the inflamed lymph-nodes and in the blood, viscera, and the various excretions.

Morbid Anatomy.—The post-mortem appearances are very similar to those of other severe infections. The blood is dark and fluid; hemorrhagic extravasations are seen in the serous and mucous membranes; the liver and spleen are swollen; there is parenchymatous degeneration of the various viscera. Many of the lymph-nodes are inflamed and some of these show foci of suppuration. Hypostatic pneumonia is common.

Symptoms.—The INCUBATION PERIOD is usually less than one week; most commonly from three to five days. The onset is sudden, usually without prodromata, and consists of headache, malaise, fever and great muscular weakness. A rigor is not common.

The disease appears in four forms. Of these the ordinary or *severe type* (*pestis major*) is marked by high fever (104°–105° F.), great prostration, anxious look, rapid pulse, dry, brown tongue, and, after three days, by the appearance of glandular swellings (buboes) which may reach the size of an egg and which usually suppurate and break down. These buboes are present in about three-fourths of the cases and most frequently occur in the inguinal glands. Less commonly they develop in the axillary or cervical glands. A tendency to hemorrhage from the mucous membranes and into the skin is common in the later days of the disease.

(2) The *mild form* (*pestis minor*) is seen chiefly at the beginning of an epidemic and is characterized by symptoms of only moderate severity and by the absence of glandular swellings.

(3) The *malignant type* (*pestis siderans*) is marked by symptoms of an intense septicemia which is usually fatal within a day or two and before buboes have had time to develop.

(4) The *pneumonic type* presents the symptoms and signs of severe pneumonia. The recent Manchurian outbreak was wholly pneumonic in type and invariably fatal.

Prognosis.—The mortality ranges between 50 and 90 per cent. The malignant and pneumonic cases are almost uniformly fatal, as are also many of the cases of the common bubonic type.

Diagnosis.—Cultures from the local lesions or from the blood regularly yield the characteristic organism.

Treatment.—Prophylaxis demands careful isolation and the disinfection of all discharges, clothing, infected houses, etc. Special attention should be paid to the destruction of rats and to the protection of exposed persons from the bites of fleas and other insects. As the pneumonic type is probably conveyed from one person to another by inhalation, physicians and other workers in the Manchurian epidemics were protected by a respirator consisting of antiseptic cotton between layers of gauze, as well as a head dress, overalls, and rubber gloves.

Serum Treatment.—Haffkine's preventive serum seems to confer a fair amount of immunity for a few weeks and to mitigate the severity of the symptoms in those attacked. The value of Yersin's curative serum seems not yet to be fully established.

Lustig and Galeotti dissolve agar-agar plate cultures of the bacillus in 1 per cent. caustic potash solution, precipitate the nucleoproteids by the addition of weak acetic or hydrochloric acid, wash and dry the precipitate in vacuo. Three milligrammes of this substance dissolved in 0.5 per cent. carbonate of soda are given by injection. A marked local and general reaction follows the inoculation, the reaction becoming less with each repetition of the injection. Favorable results have been reported from the use of these inoculations in man. By treating the horse with these injections a serum which is both protective and curative in animals has been obtained. Otherwise the treatment of the disease is purely symptomatic.

MALTA FEVER

(Undulant Fever. Mediterranean Fever)

Definition.—Malta fever is a specific infectious disease of protracted febrile course endemic in Malta, Gibraltar and the regions bordering on the Mediterranean. A few cases have been met with in other parts of the world. It attacks young adults chiefly and is most prevalent in summer. The specific agent is the *Micrococcus melitensis* (Bruce), which is found abundantly in the spleen and lymph-nodes and rarely in the blood. The disease is not contagious. The fever is spread in Malta through the common use of goat's milk. Fifty per cent. of the goats on the island are infected.

Morbid Anatomy.—Fatal cases show no characteristic lesions. The spleen is much enlarged and soft and there is sometimes a complicating pneumonia. The organism can be cultivated from the spleen and lymph-nodes.

Symptoms.—The onset is slow and resembles closely that of typhoid fever. The disease runs a very protracted course of from two to six (or more) months, made up of a series of febrile waves, each lasting from one to three weeks. The fever rises and subsides gradually and the waves are separated by a few days' interval of normal or nearly normal temperature. In some cases these undulations are not present and there is a continued fever with daily remissions or intermissions. Gradual emaciation, weakness and anemia follow, but delirium and severe toxic symptoms are rare. Constipation, profuse sweating and neuralgic pains are common. Almost the only physical sign is the constant great enlargement of the spleen.

Inflammations of the joints and testicles are frequent complications.

Diagnosis.—From typhoid the disease may be distinguished by the peculiar course of the temperature and by the absence of an eruption, of diarrhea and of the Widal reaction and by the results of blood cultures. Moreover, the blood-serum, even when highly diluted, causes the prompt agglutinization of cultures of the micrococcus melitensis, and this organism can be isolated from the splenic blood obtained by puncture.

Prognosis.—Almost all the cases recover in spite of the greatly prolonged and tedious course, the mortality being only 2 per cent. The rare fatal cases usually run a short course with very high fever and severe toxic symptoms.

Treatment.—The treatment is purely symptomatic.

BERI-BERI

Definition.—Beri-beri is an endemic and epidemic disease of warm countries, probably infectious in nature, characterized by a neuritis affecting chiefly the peripheral, cardiac and vasomotor nerves.

Etiology.—The disease prevails in many tropical and sub-tropical regions, especially in Japan, China, the East Indies and South America. A few epidemics have occurred in asylums in our Southern States and cases have been met with among the Gloucester fishermen. It exists endemically in many regions and from these may spread from time to time in the form of epidemics. It attacks chiefly the native population and is prone to appear in crowded, damp and unsanitary ships, asylums, barracks, etc. It is favored by hot, damp weather. Young adult males are those most frequently attacked.

Two theories are held concerning its causation. The one more generally accepted is that it is a specific infectious disease. The other view regards the disease as a species of toxemia due to the eating of certain kinds of food, especially of diseased rice or of raw fish. No germ has yet been proven to be the exciting cause. The disease is not directly contagious.

Morbid Anatomy.—The characteristic lesion is that of a neuritis with degeneration of the nerve fibers and of the associated muscles.

When the vagus is involved the heart is frequently much dilated and shows parenchymatous degeneration.

Symptoms.—These vary much both in character and in severity. When the nerves of the extremities are chiefly affected the clinical picture is very similar to that of alcoholic neuritis. When, as often happens, the cardiac and vasomotor nerves are involved cardiac disturbance, dyspnea and anasarca are the conspicuous symptoms. Four clinical types may be recognized:

(1) A mild or rudimentary form in which, with or without vague prodromal symptoms, there appear weakness and pains in the legs, tenderness of the calf muscles, dulling of tactile sensation or paresthesia of the arms and legs, slight edema and some breathlessness and cardiac irritability. These symptoms may last for weeks or months.

(2) *The Atrophic or Dry Form.*—In this there are marked loss of power in the arms and legs, much wasting of the muscles, loss of the deep reflexes, tenderness of the muscles, pain, and anesthesia or hyperesthesia of the skin. Cardiac symptoms and dropsy are usually slight or lacking, but death sometimes occurs from sudden heart failure. Most cases recover after many months of illness.

(3) *The Dropsical or Wet Form.*—Dropsy is a conspicuous feature. There are marked edema of the subcutaneous tissue and effusion into the serous cavities. The heart action is weak and often irregular and there is much dyspnea. The evidences of peripheral neuritis are relatively slight.

(4) *The Acute, Pernicious Form.*—This is a very fatal type which may run its course in a day or two, but which usually lasts for several weeks. There are grave disturbance of the heart action and great dyspnea, which may be constant, but is apt to appear in paroxysms of alarming severity. Involvement of the phrenic nerve may cause paralysis of the diaphragm. The heart is usually much dilated. Death usually results suddenly from cardiac failure.

Prognosis.—The mortality varies in different epidemics from three to fifty per cent. The prognosis is always uncertain, since even in the apparently mild cases sudden death may result from vagus neuritis.

Treatment.—Whenever possible the patient should be at once removed from the infected region and placed under the best hygienic conditions. An abundance of nitrogenous food is required and rice should be avoided. Absolute rest in bed and cardiac stimulants are demanded if the heart symptoms are pronounced. For the paralyzed muscles galvanism and massage are often of value.

SCARLET FEVER

(Scarlatina)

Definition.—Scarlet fever is an acute, contagious disease characterized by an abrupt onset, fever, sore throat, and a diffuse erythematous rash.

Etiology.—The disease is one of the common exanthemata of child-hood and is found scattered widely over the whole world. It is endemic in most cities, and in less thickly populated districts occurs sporadically and in small or moderate sized epidemics. It attacks chiefly children between the ages of two and ten years and is rare in infancy and in adult life. The sexes are equally susceptible. Most of the cases occur in the colder months of the year.

Scarlet fever is only moderately contagious. Not more than half of the children exposed to the disease contract it. It is contagious from the onset of symptoms to the end of desquamation. Of recent years less importance has been given desquamation and more emphasis put upon discharges from the throat, nose, or ears. The contagium is given off from the skin, in the discharges from the nose, throat and ear, and probably in the urine and feces. The disease is transmitted by direct contact; by means of a third person (nurse, physician, etc.), or by fomites (clothing, toys, carpets, etc.). The poison is notably resistant and may remain active in a room, or in clothing, for months or even for a year or more. Recently a few epidemics have been traced to contaminated milk.

The germ of scarlet fever is still unknown. The streptococcus pyogenes is the bacterium usually found in the severe throat and ear complications.

One attack almost always confers immunity for life.

Morbid Anatomy.—The only essential lesions are those of the skin and throat. There is a severe acute dermatitis, as shown by intense hyperemia, exudation of round cells and edematous swelling, which results in the death of the epidermis and its desquamation. In the throat there is usually a severe catarrhal pharyngitis. The other lesions found at autopsy will be mentioned under Complications.

Symptoms.—The INCUBATION PERIOD is regularly less than one week. Most commonly it is three or four days. It may, however, be less than 24 hours.

INVASION.—The onset is almost always sudden, with *vomiting, prostration, sore throat* and *fever.* In young children a convulsion is not uncommon. The temperature rises rapidly and is directly proportionate to the severity of the case. In severe cases it may reach 104°–105° F. by the end of the first day. The skin is dry and hot, the pulse very rapid, the tongue coated and the pharynx and fauces usually show a diffuse reddening.

ERUPTION.—Between 12 and 36 hours after the first symptoms there appears upon the neck and chest a rash composed of minute, red, punctate spots which rapidly spreads over trunk, extremities and face and becomes an intense, diffuse scarlet flush. The lips and chin usually escape and show a pallor in striking contrast to the appearance of the rest of the body. In mild cases the eruption may be faint and may not be seen at all upon the face. Occasionally, too, it is not uniform and diffuse, but occurs in large blotches or in macular spots resembling

measles. The rash remains distinct for two or three days and then gradually fades, the whole duration being from three to seven days. (See Fig. 67.)

While the rash is distinct the fever remains high and there is much burning and itching of the inflamed skin. The child may be either very restless or stuporous. Often there is active delirium. The severity of the throat symptoms varies greatly. Usually there are redness and swelling of the pharynx, tonsils, fauces and uvula, with some pain on swallowing and some swelling and tenderness of the submaxillary lymph-nodes. The tongue at first is coated with a white fur, but later is dry and red. The papillæ are swollen and project above the surface of the dorsum, producing the so-called "strawberry tongue." With the fading of the eruption the temperature gradually falls and the other constitutional symptoms subside. The duration of the fever is commonly from seven to ten days.
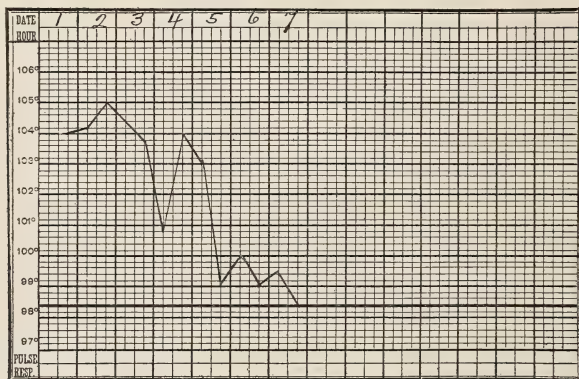
Fig. 67.—Temperature curve of uncomplicated scarlet fever.

DESQUAMATION.—As soon as the rash disappears there begins a desquamation or "peeling" of the dead epidermis, which is first noticed upon the chest and neck and extends to all parts of the body where the rash has been pronounced. Over the trunk it occurs in flakes and scales, but on the hands and feet, where it is always marked, the epidermis comes off in large strips or even as whole casts of the fingers or toes. This form of peeling is very characteristic of the disease (see Figs. 68, A and B).

The period of desquamation lasts from two to four, or even to six, weeks. The process is slowest on the hands and feet where the cuticle is thickest.

CLINICAL TYPES.—In addition to the cases of moderate severity described above there are

1. *Mild Cases.*—In these the onset is abrupt, but the temperature rises only to 102° or 103° F., and may reach normal within four or five

days. The rash may not involve the face and may be only faint and transient over the body. The throat symptoms are usually slight.

Such cases may easily be overlooked, especially when they occur in adults. They are important not only because they may transmit the disease in its severer forms, but also because they may be followed by a serious nephritis or other complication.

2. *Severe Cases.*—The period of incubation is short; the onset violent; the rash appears early, is pronounced and widespread, and the fever is high and protracted (two to three weeks). Delirium alternates with periods of stupor; the pulse grows steadily feebler and more rapid; the throat symptoms are very severe. Complications are very common and the child is likely to die either of the severe toxemia or of the complications.
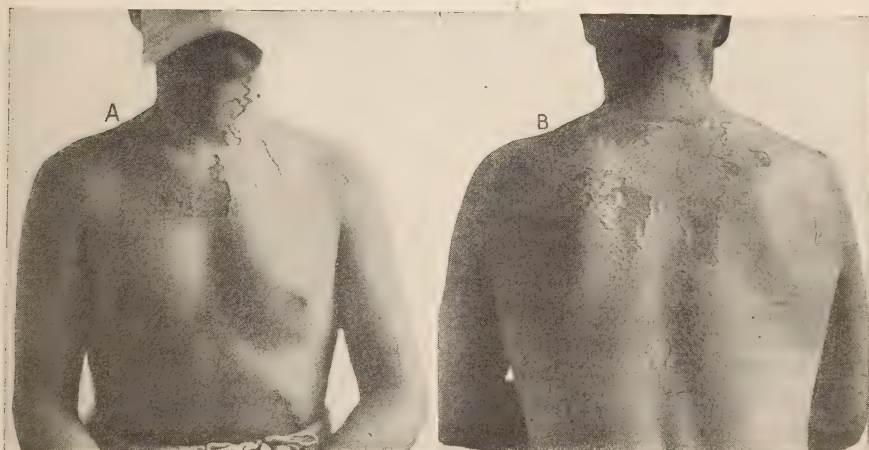
FIG. 68.—*A*, desquamation of scarlet fever on neck and chest; *B*, desquamation of scarlet fever on back.

3. *The Malignant Form.*—The onset is violent, usually with convulsions and persistent vomiting; the fever is extremely high (105°–108°), and the pulse very rapid and feeble. Delirium soon gives place to coma and the child dies within forty-eight hours, overwhelmed by the poison of the disease. The rash is faint, irregular or may be wanting altogether. Exceptionally the eruption is hemorrhagic and accompanied by bleeding from the mucous surfaces. Malignant scarlet fever is fortunately rare and is seen only during severe epidemics.

4. *The Anginose Form.*—This name is given to those distressing cases in which the throat inflammation is extremely severe. There is from the outset rigidity and tenderness of the neck and jaws and pain and difficulty in swallowing. The pharynx, fauces, tonsils and uvula are intensely red and upon the third or fourth day show a membranous exudate which may extend into the posterior nares or larynx. There is an offensive, sanious discharge from the nose and mouth; the cervical

glands are greatly swollen, and there is often a diffuse cellulitis of the neck. Necrosis and sloughing of the tonsils or soft palate or cervical glands may occur. The constitutional disturbance is extreme and the cases are usually fatal.

SURGICAL SCARLET FEVER.—While it is undoubtedly true that puerperal women and persons with fresh wounds are peculiarly susceptible to scarlet fever, it is also true that many of the fevers seen in these cases are not true scarlet fever, but septicemia with an erythematous rash.

RELAPSES.—A recrudescence of the fever, eruption, angina and other symptoms is occasionally seen during convalescence.

Complications and Sequelæ.—These are numerous and very important.

NEPHRITIS.—A slight albuminuria is seen in most of the cases during the active stage and has little significance. True acute nephritis may occur at any time, but is usually seen during desquamation. It may occur as late as the sixth week. All grades of severity are met with. The mild cases recover promptly. In the severer forms many of the children die of uremia or else the acute symptoms subside and the disease persists as a chronic nephritis.

OTITIS MEDIA.—Suppurative inflammation of the middle ear, due to infection through the Eustachian tube, is a common and important complication, since it may result in permanent deafness or in mastoiditis, thrombosis of the lateral sinus, meningitis or brain abscess. Its frequency bears a direct relation to the severity of the throat inflammation. It is often bilateral.

ARTHRITIS.—A synovitis, serous or purulent, is occasionally seen. Usually it affects several large joints and closely simulates acute articular rheumatism.

THE HEART.—Simple acute endocarditis, pericarditis (serous or purulent) and acute myocarditis are occasional complications. The former often results in permanent deformity of the valves.

THE LUNGS.—Bronchopneumonia is found in most of the fatal cases. Pleurisy with effusion and empyema are both not uncommon.

CELLULITIS OF THE NECK is a grave complication, seen especially with the anginose form. The abscess usually begins in the cervical lymph-nodes.

TRUE DIPHTHERIA sometimes develops during convalescence. The membranous angina so frequently seen, however, is usually associated with the streptococcus pyogenes and is therefore not true diphtheria.

Diagnosis.—Confusion is likely to occur only in atypical cases such as those of the malignant form, in which the rash is irregular or is lacking, and of the anginose form, which may readily be mistaken for diphtheria. The diagnosis is to be made quite as much by the abrupt onset, the vomiting, the rapid rise of temperature and the very early appearance of the rash as by the character of the rash itself. This may be simulated by a number of other rashes, especially by those of septicemia, urticaria, measles, rubella, acute exfoliative dermatitis and by

PLATE II.

A   B

C   D

THE PATHOGNOMONIC SIGN OF MEASLES (KOPLIK'S SPOTS).

 A. The discrete measles spots on the buccal or labial mucous membranes, showing the isolated rose-red spot, with the minute bluish-white centre, on the normally colored mucous membrane.

 B. Shows the partially diffuse eruption on the mucous membrane of the cheeks and lips; patches of pale pink interspersed among rose-red patches, the latter showing numerous pale bluish-white spots.

 C. The appearance of the buccal or labial mucous membrane when the measles spots completely coalesce and give a diffuse redness, with the myriads of bluish-white specks. The exanthema on the skin is at this time generally fully developed.

 D. Aphthous stomatitis apt to be mistaken for measles spots. Mucous membrane normal in line. Minute *yellow points* are surrounded by a red area. Always discrete.—*Med. News*, June 3, 1899.

those caused by certain drugs, such as quinine, sodium salicylate, bella-donna, potassium iodide and antipyrine. In all of these conditions there are lacking the characteristic onset and early symptoms of scarlet fever, and the universal distribution of the rash.

Prognosis.—The average mortality is from 10 to 15 per cent. It varies, however, in different epidemics between five and forty per cent. The disease is especially fatal in children under two years. Death may be caused by the intense systemic poisoning, by sepsis from the severe angina, by such complications as bronchopneumonia and severe otitis media or by post-scarlatinal nephritis.

Prophylaxis.—The child should be immediately isolated and no one but the nurse and physician should enter the sick-room. Other children in the family should be at once sent away. The three chief sources of contagion are the patient, the nurse, and the room. The patient will usually have to be kept in his room for six weeks. If desquamation is not then complete, or if there still persist discharges from the nose, throat, ear or from abscesses, he must be isolated until these have ceased. The danger from desquamation can be diminished by keeping the skin anointed with carbolized vaseline or some bland ointment. Before he leaves the sick-room every part of the child's body should be thoroughly scrubbed with soap and water several times and sponged with a 1–5000 solution of corrosive sublimate. The urine and feces are to be disinfected as described under Typhoid Fever. Discharges from nose, ears, abscesses, etc., should be received into surgical gauze or absorbent cotton, which can be burned. Soiled linen is soaked for one hour at least in a 1–20 solution of carbolic acid and afterward boiled for two hours in water. (See also Appendix, p. 583.)

The nurse should be kept apart from the rest of the family as much as possible and should change her clothing and disinfect her hands and face upon leaving the sick-room. The physician should wear in the room a gown and head covering.

Before the sick-room is occupied it should, if possible, be stripped of its carpet, hangings and upholstered furniture. After the patient has left the room it should receive most thorough disinfection. The walls if painted can be washed with bichloride solution, otherwise they are best cleaned by being rubbed down with moist bread, the fragments of which can be burned. The floor and woodwork are scrubbed with a solution of bichloride of mercury 1–1000. If possible books and toys should be burned. Clothing, carpets, mattress, upholstered furniture are best disinfected in the steam disinfecting stations provided by most large cities. If this is not feasible, exposure to direct sunlight for a number of days is probably the most useful measure. The method so much in vogue of simply fumigating the room and all its contents with sulphur is of exceedingly doubtful value. Formaldehyde gas is more trustworthy, but to be effective must be used in larger quantities than most of the lamps now on sale will generate.

Treatment.—This consists in careful nursing, the prevention of

complications and the combating of individual symptoms. The child is at once put to bed and kept there until the temperature has been normal for a week. A fluid diet (chiefly milk) is maintained for the same period. The room must be well ventilated, but great care should be exercised to prevent the patient from taking cold. The child should be made to drink very freely of water throughout the whole of the disease. The danger of nephritis can be further diminished by the moderate use of saline laxatives and by keeping the skin active by means of warm baths. The urine should be examined frequently.

The nose and throat are kept clean by free irrigation with some mild antiseptic or with normal salt solution. If the throat symptoms be severe, however, this is of little avail in preventing otitis. The burning and itching of the rash can be somewhat relieved by keeping the skin well protected by vaseline or boric acid ointment.

Hyperpyrexia and the severe nervous symptoms are best treated by warm tub baths, which can be gradually cooled to 85° F. or less as indicated, or by packs. An ice-cap to the head is useful when the cerebral symptoms are marked.

In severe cases when the heart weakness threatens life stimulants must be used freely. Of these alcohol, in the form of brandy or whiskey well diluted, is perhaps the best. It is usually well borne and can be given in relatively large amounts (as much as three ounces in twenty-four hours to a child of four years). Strychnine and digitalis are also valuable. The local treatment of the throat in the anginose form is that described under Diphtheria.

If otitis media develop, the drum membrane must be watched closely and puncture resorted to if the accumulation of fluid be marked.

Nephritis, if it appear, is to be treated as though it were a primary condition. (See p. 160.)

Suppuration of the lymph-glands or the cellular tissue of the neck demands immediate incision.

MEASLES
(Morbilli.   Rubeola)

Definition.—Measles is an acute, highly contagious disease of childhood which manifests itself by coryza, conjunctivitis, moderate fever and a diffuse rose-red maculopapular eruption.

Etiology.—The disease is one of the most widespread and highly contagious known. It is transmitted usually by direct exposure; conveyance by means of a third person or by fomites being much less common than in scarlet fever. Susceptibility is almost universal in children, but the disease is rarely seen in infants of less than six months. It may occur in adults. Most of the epidemics occur during the colder months of the year. The contagious period extends from the beginning of the catarrhal symptoms to the end of desquamation, the contagiousness being very pronounced even before the appearance of the eruption. Protection is not always secured by one attack.

PLATE III.

The Eruptions of Measles and Scarlet Fever.

Scarlet Fever.

Measles.

Morbid Anatomy.—The only lesions constantly present are those of the cutaneous eruption and the catarrhal inflammation of the nose, conjunctivæ, larynx, trachea and large bronchi. Bronchopneumonia is regularly present in the fatal cases.

Symptoms.—The INCUBATION occupies quite regularly from ten to fourteen days.

INVASION.—The onset is usually gradual, the symptoms being those of a severe cold; sneezing, catarrhal rhinitis, suffused and reddened eyes, hoarseness and hoarse cough. The temperature rises within two or three days to 103° or 104° F. and the child is fretful, listless and drowsy. Not infrequently the temperature, instead of rising steadily, will fall on the second or third day and rise again with the appearance of the rash. (See Fig. 69.) Occasionally the onset is more abrupt and violent, but a distinct chill is rare. The stage of invasion lasts from two to four days.
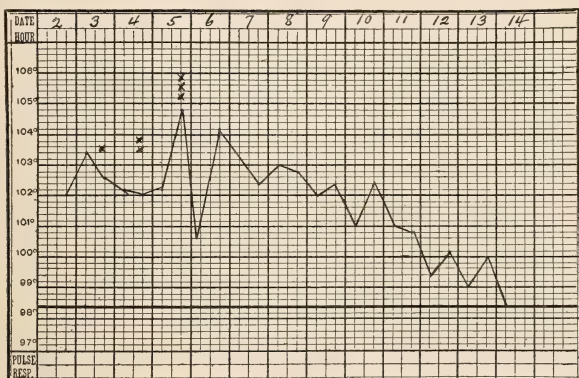
FIG. 69.—Temperature curve of severe measles. The crosses mark the development of the eruption. One day of slight fever preceded the period charted.

ERUPTION.—The rash appears on the third or fourth day and shows first on the forehead, neck and behind the ears. In many cases on the day before the eruption small red spots with bluish-white centers may be seen scattered over the mucosa of the palate and cheeks (Koplik's spots) (see Plate II). The rash at first consists of a few small, macular spots of a deep rose-red color, which soon change to soft, velvety papules and become much more numerous, spreading slowly over the face, trunk, arms and legs in the order named and occupying three or four days in the process. The papules arrange themselves in large groups which are apt to take a crescentic or rounded outline and many of which fuse into diffuse large blotches (see Plate III). By the time the rash appears on the legs it has begun to fade upon the face. Meanwhile the fever and all the catarrhal symptoms have persisted and there are intense itching and burning of the skin and often diarrhea. With the fading of the rash the temperature falls rapidly and the other symptoms gradually subside.

DESQUAMATION begins soon after the rash has disappeared (usually on the eighth or ninth day) and lasts from five to ten days. It is always fine and bran-like (furfuraceous) in character.

Not all the cases run the typical course described. There are MILD CASES in which the fever is slight and the rash and the catarrhal symptoms less intense; and SEVERE CASES in which the fever is high and protracted and which are usually complicated by bronchopneumonia. MALIGNANT CASES, in which the child is overpowered by the intensity of the poison, are fortunately rare. There is also a severe form called "black measles" marked by a dark colored and hemorrhagic rash and often by bleeding from the mucous membranes.

Complications and Sequelæ.—The chief complications are bronchopneumonia, pseudo-membranous laryngitis and pharyngitis, otitis media, pleurisy, enterocolitis, and ulcerative or gangrenous stomatitis. Nephritis is rare. Of the sequelæ tuberculosis (general, pulmonary or glandular) is much the most important. Chronic conjunctivitis and chronic enlargement of the lymph-glands are not uncommon.

Diagnosis.—The chief differential points in distinguishing measles from the other exanthemata are the gradual onset, the marked catarrhal symptoms, the late appearance of the rash, its slow development and its large blotches with crescentic outlines. The Koplik spots upon the palate and buccal mucous membranes are also very suggestive.

Prognosis.—Fatal cases are rarely seen in private practice among children over three years. Below this age the mortality is about ten per cent. In asylums and in tenements, however, the death rate is often much higher. Bronchopneumonia is the commonest cause of death.

Treatment.—PROPHYLAXIS.—Early isolation of the patient is most important. Children who have been exposed should be kept away from other children for sixteen days. The patient himself must be isolated for at least three weeks. The child should be kept in bed, and upon fluid diet, throughout the febrile period, and the room kept darkened and well ventilated. The eyes are to be kept clean by frequent bathing with a saturated solution of boric acid. The itching of the eruption may be lessened by the application of some bland ointment. During desquamation daily warm baths should be given. Troublesome cough may be relieved by ipecac and paregoric or by small doses of codeine (gr. 1/15–1/30). If the bronchitis be severe, counterirritation in the form of mustard pastes frequently applied to the chest is often of great value. The treatment of persistently high temperature, of severe nervous symptoms and of heart weakness is that described under Scarlet Fever. During convalescence great care is needed, not only to prevent the complications, but also to improve as rapidly as possible the child's physical condition, in the hope of thereby diminishing the chance of bronchopneumonia and tuberculosis. Nutritious food, tonics, cod liver oil, etc., are needed and a change of climate is often desirable.

RUBELLA

(German Measles. Rötheln)

Definition.—Rubella is an acute contagious disorder characterized by a pronounced but variable rash, slight constitutional disturbance and the absence of complications. It was formerly much confused with measles and scarlet fever, from both of which, however, it is entirely distinct.

The disease is only moderately contagious, occurs in epidemics and attacks adults as well as children.

Symptoms.—The INCUBATION PERIOD varies much, but is commonly from ten to fifteen days.

In many cases no symptoms whatever are noticed before the rash appears. Usually, however, for a few hours before the eruption is seen, there are some headache, malaise, slight fever and perhaps a mild coryza. This stage of invasion is almost always less than two days.

ERUPTION.—The rash often closely resembles that of measles; less frequently it is scarlatiniform. It appears first upon the face, spreads over the whole body within 24 hours, persists for two or three days and then fades rapidly. It is composed usually of minute pale red, slightly elevated spots, which, upon the face, may become confluent. Desquamation is very slight or altogether lacking. With the rash the temperature may reach 101° or 102° F.; there is often a mild sore throat, some itching of the skin, and the posterior cervical lymph-glands are regularly swollen. Complications are almost unknown and the prognosis is uniformly good.

Diagnosis.—It is frequently impossible, by the rash alone, to distinguish German measles from true measles or from scarlatina. It differs from the former, however, in its very mild course, the absence of marked catarrhal symptoms, the early appearance of the rash, the absence of Koplik's spots, the swelling of the lymph-glands and the absence of complications; and from scarlet fever in its long incubation, its very mild onset and course, its mild throat symptoms, the glandular enlargement and its freedom from desquamation.

No treatment but good nursing is required. It is necessary to insist upon isolation.

DIPHTHERIA

Definition.—An acute contagious disease caused by a specific micro-organism—the *bacillus diphtheriæ*, and marked, usually, by a pseudo-membranous inflammation of the fauces, pharynx, nose or larynx and by constitutional symptoms due to the absorption of toxins.

Careful bacteriological studies of diphtheria and its allied affections have demonstrated, first, that the *bacillus diphtheriæ* does not always excite an inflammation with the production of a distinct false membrane, but may occasionally cause only a slight catarrhal process; and second, that such pseudo-membranous inflammations are sometimes caused by

other bacteria than the diphtheria bacillus. The presence of this organism then, in any given case, is the only certain evidence that the disease is true diphtheria.

For such pseudo-membranous inflammations as are caused by other bacteria Osler has suggested the term *diphtheroid*.

Etiology.—Diphtheria has long been known as one of the common and important contagious diseases, and occurs endemically, epidemically and sporadically. It is endemic in most large cities, where from time to time also the cases multiply to such an extent as to constitute an epidemic. In the country it is seen chiefly in small epidemics.

PREDISPOSING CAUSES.—The disease is widely distributed throughout the world. It occurs at all seasons, but is most prevalent during the winter months. Children under ten years are much more liable to infection than are adults; the age of greatest susceptibility being from two to five years. Infants are rarely attacked. The disease is slightly commoner in girls than in boys. Predisposition to the disease is greatly increased by such abnormal conditions in the throat as enlarged tonsils, adenoid growths or chronic naso-pharyngeal catarrh. The occasional association of true diphtheria with such diseases as scarlet fever and measles is probably to be explained in this way.

The EXCITING CAUSE is the *bacillus diphtheriæ,* known also from its discoverers (1883–1884) as the Klebs-Löffler bacillus. This is a non-motile, slender rod of medium length, straight or slightly bent and with rounded and often club-shaped ends. It stains peculiarly in that different parts of the organism often stain with different intensity and so give to it a somewhat beaded appearance. This tendency to variability in shape and staining is a feature of much practical value for the identification of the organisms. The bacilli grow most readily upon a medium composed chiefly of blood serum and, at body temperature, the colonies become visible within 10 or 12 hours. The Klebs-Löffler bacillus is very tenacious of life and may live for many weeks or months even under unfavorable conditions. The organisms are always present in the throats of those ill of the disease and are especially numerous in the superficial layers of the false membrane. In the deeper parts of the mucous membrane and in the circulating blood they are but rarely found.

TRANSMISSION.—Diphtheria is transmitted only by means of these bacilli and every case has its origin in some other. The manner of such communication is, however, often very obscure and difficult to trace. The bacilli are given off in the secretions and discharges of the mouth, throat and nose and especially in the fragments of false membrane. Such discharges reach the mouth and throat of other persons either directly, as in the act of coughing or kissing, or indirectly through the medium of toys, bedding, clothing, dishes, etc. The organism thrives in milk and certain epidemics have been traced to this source. Drinking water is believed never to carry the contagion. Physicians and nurses in attendance upon cases of diphtheria are frequently infected, and

even when not ill, their throats have occasionally been found to contain diphtheria bacilli. In patients convalescent from the disease, the bacilli sometimes remain in the throat and retain their virulence for several weeks or even longer, and such persons probably play an important part in the dissemination of the disease. The same is true of those cases of diphtheria which develop little or no pseudo-membrane and which are therefore often mistaken for simple sore throat. Cats are subject to a similar pseudo-membranous affection and diphtheria may perhaps sometimes be conveyed in that way, although the identity of the two diseases has not yet been thoroughly established. Sewer gas and defective drainage are no longer regarded as important causative agents. Although second attacks are not common the disease does not confer the same degree of immunity as do most of the other contagious diseases.

Toxin.—The diphtheria bacillus in its growth produces a chemical poison—a toxin—which is readily absorbed from the site of the local lesion and is carried throughout the body by the blood. Most of the constitutional symptoms and severe visceral lesions have been shown to be due to the action of this toxin.

Other bacteria are frequently found associated in the throat with Klebs-Löffler bacillus. Of these the STREPTOCOCCUS PYOGENES is the most important. To it are due certain secondary suppurative processes and septicemias.

Morbid Anatomy.—The diphtheria bacilli, in the vast majority of cases, find lodgment upon, and excite an inflammation of, the mucous membrane of the upper air passages. In rare instances, however, the false membrane may be seen in the stomach, middle ear, conjunctivæ, vagina or upon open wounds. The membrane most frequently appears upon one or both tonsils and from these often spreads over the soft palate and uvula, and back to the lateral and posterior walls of the pharynx. It frequently extends also into the naso-pharynx and from there into the posterior nares and nose. In other cases the extension is downward to the epiglottis, larynx, trachea and bronchi. Not infrequently the process begins in the nose or in the larynx.

The false membrane differs greatly in color, extent and consistence in different cases and at different stages of the same case. The color is usually a dirty gray or yellow, but may be greenish, brown or almost white. The membrane may be thick, dense and firmly adherent or loose, friable and shreddy. Occasionally the inflammation is marked by extensive sloughing not only of the mucous membrane but of the deeper structure as well, so that even large blood-vessels may be eroded and the tissues of the neck become the seat of extensive and deep-seated suppuration. This grave condition is especially likely to occur when other bacteria, such as the streptococcus, are acting in conjunction with the diphtheria bacillus.

Histologically the process is an acute exudative inflammation with necrosis of the epithelial cells of the superficial layers of the mucous membrane. The false membrane consists of a meshwork of fibrin

enclosing leukocytes, red cells and masses of dead epithelial cells. These dead cells undergo a peculiar hyaline transformation (coagulation necrosis), and constitute an essential feature of the process.

OTHER LESIONS.—Post-mortem examination shows that the cell necrosis is by no means confined to the mucous membrane. It is produced by the toxin rather than by the direct action of the bacilli, and is often found in the cervical lymph-nodes, in the parenchyma of the heart, kidneys and liver and in the peripheral nerves. In fatal laryngeal cases the lungs always show areas of atelectasis and of bronchopneumonia.

Symptoms.—The clinical picture of diphtheria varies greatly, and depends not only upon the site and severity of the local process, but also upon the amount of toxin produced and absorbed. It is to the constitutional effects of this toxin that many of the gravest symptoms are due.

The INCUBATION PERIOD is rarely longer than one week and usually lasts only from two to five days.

INVASION.—This is apt to be mild and insidious rather than abrupt and violent, although exceptions to this rule are common. In very young children the onset is frequently abrupt, with a sudden rise of temperature, vomiting or convulsions. Usually, however, headache, malaise, loss of appetite, slight fever and slight sore throat mark the beginning of the disease.

1. PHARYNGEAL DIPHTHERIA.—*Mild Type.*—The membrane appears upon the tonsils and is often confined to them, being usually small in amount. It may, however, extend somewhat over the faucial pillars or the lateral pharyngeal wall. The onset is mild and the constitutional symptoms slight. The fever ranges between 100° and 102° F. The throat feels uncomfortable, there is some pain on swallowing, the lymphglands below the angle of the jaw are tender and swollen. The child may not feel sick enough to be in bed. The throat looks somewhat red and swollen; the membrane at first may be only a film or may be made up of several small white spots closely resembling those of simple follicular tonsillitis. When well developed it forms a gray or whitish patch firmly adherent to the mucosa. If forcibly detached it leaves a bleeding surface, which is promptly re-covered by false membrane. The membrane lasts from three days to a week or more. By the time of its disappearance the child is usually convalescent. Complications are infrequent. This type is frequent among older children and adults.

Severe Type.—The symptoms at first are similar to those of the mild form, but the membrane shows a tendency to extend, and soon covers the fauces, uvula and pharyngeal wall. Often, too, it spreads to the posterior nares or to the larynx. The tonsils and uvula are much swollen and there is a free secretion of mucus. Although usually the temperature is not high (100°–103° F.), the constitutional symptoms soon become marked. The prostration is extreme; the pulse grows progressively weaker and more rapid (110–140); there is apathy, stupor

or great restlessness; pallor is pronounced; there may be vomiting or diarrhea. The blood shows a rapidly progressive anemia and usually a well-marked leukocytosis; the cervical lymph-glands are much swollen; the urine is albuminous and often contains casts and blood cells. In rare instances the patient is fairly overwhelmed by the intensity of the poisons and dies within two or three days. Usually the toxemia is most pronounced from the fifth to the tenth day. Death is common during this period and not infrequently comes suddenly and unexpectedly. It is usually attributed to heart failure from myocardial degeneration or from neuritis of the cardiac nerves. Recent studies have seemed to show, however, that this collapse is due primarily to paralysis of the vaso-motor control of the vessels rather than to derangement of the heart itself. If the patient survives, the membrane begins to loosen and come away during the second week, and convalescence, often slow and tedious, begins.

A distressing and grave form of the disease is that known as *septic diphtheria*, in which the local symptoms are especially severe (being due to a mixed infection by the diphtheria bacillus and the *streptococcus pyogenes*), and in which evidences of septicemia or pyemia develop.

Cases Without a Membrane.—A small but important class of cases are those which show only a catarrhal inflammation of the throat, with no membrane whatever, or with only a few white spots upon the tonsils which cannot be distinguished from those of simple follicular tonsillitis. And yet these are cases of true diphtheria, are liable to the usual complications of the disease and are capable of spreading contagion.

2. NASAL DIPHTHERIA.—The infection may be primary in the nose, but it usually occurs by extension from the naso-pharynx. The membrane may completely fill the nose and may be seen at the anterior nares. The local symptoms are those of obstruction of the nares, together with a nasal discharge which is usually profuse, thin, acrid and often blood tinged. The constitutional symptoms are as a rule very severe and the glandular swelling marked. There may be an extension of the membrane through the Eustachian tube to the middle ear or through the lachrymal duct to the conjunctiva.

3. LARYNGEAL DIPHTHERIA.—(*Membranous croup*).—It is fully established that membranous laryngitis, or croup, is in the vast majority of cases true diphtheria. The chief exceptions to this rule are those cases which develop in the course of such diseases as scarlet fever and measles and which are usually due to the *streptococcus pyogenes*. The membrane commonly reaches the larynx by extension from the pharynx. Less frequently the process begins in the larynx. It frequently extends downward along the trachea and may even invade the finer subdivisions of the bronchial tree.

The laryngeal symptoms begin rather gradually with hoarseness or whispering voice, a harsh, "croupy" cough and a moderate rise of temperature. The breathing gradually becomes more labored and within

a day or two the symptoms of laryngeal stenosis are usually well marked. Each inspiration is accompanied by a stridulous sound, by dilatation of the nostrils and by a sinking in of the tissues of the neck and of the epigastrium. Expiration also is difficult and sometimes stridulous. The child struggles for breath, becomes pale and then cyanotic and finally, in many cases, dies of suffocation within two or three days. If the larynx alone is involved the toxemic symptoms are usually slight. In favorable cases the obstruction is not severe and after from two to five days the membrane loosens and is coughed up and the child goes on to recovery.

Complications and Sequelæ.—BRONCHOPNEUMONIA is very common, especially in cases with involvement of the larynx and in septic diphtheria. ACUTE NEPHRITIS is also frequent. TOXIC MYOCARDITIS is doubtless responsible for certain of the instances of heart failure. SUPPURATION OF CERVICAL GLANDS may occur, especially in the type of septic diphtheria. OTITIS MEDIA is occasionally met with, and such skin eruptions as erythema and urticaria are not very uncommon. Other complications are rare. The chief sequel to diphtheria is PARALYSIS. This is due to local or multiple *neuritis* caused by the diphtheria toxin, and is met with in from 10 to 15 per cent. of the cases. The paralysis usually shows itself during convalescence. It may appear as early as the end of the first week or as late as the sixth week.

The commonest form of paralysis is that of the soft palate. It is recognized by a nasal quality of the voice, by the regurgitation of fluids through the nose during the act of swallowing and by the immobility of the palate during phonation, etc. It is usually associated with anesthesia of the parts and sometimes with paralysis of the muscles of the pharynx or larynx. There may be paralysis of the eye muscles, both intrinsic and extrinsic, with the resulting loss of accommodation, strabismus, ptosis, etc. Paralysis of the heart may be sudden or may develop somewhat gradually with the symptoms of weakness, syncope, pallor and a feeble and irregular pulse which is either abnormally slow or very rapid. It is frequently fatal. Disturbance of respiration is usually due to paralysis of the diaphragm. The breathing is labored and of the costal type and there may be distressing, even fatal paroxysms of dyspnea and cyanosis.

MULTIPLE NEURITIS is not uncommon and shows itself by paralysis of the extremities, loss of tendon reflexes and disturbances of sensation. Marked ataxia may be a feature even when the actual paralysis is slight. In general both local and multiple neuritides are of short duration and recovery usually takes place within a few weeks or months.

Diagnosis.—While in most cases a careful weighing of the clinical evidence alone will suffice to make a correct diagnosis, there are nevertheless many doubtful and obscure cases which can only be cleared up by bacteriological examination. This is especially true of cases in which there is little or no membrane; of inflammations primary in the nose or larynx, and of throat affections associated with scarlet fever

and measles. The CLINICAL DIAGNOSIS is based upon both the constitutional symptoms and the local appearances. The constitutional symptoms in diphtheria are apt to begin gradually, to be mild at first and to be most pronounced several days after the onset. In follicular tonsillitis and in diphtheroid affections the onset is usually more abrupt and severe and the general symptoms of shorter duration. Of the local signs, those which point most strongly to diphtheria are the uniform, firm character of the membrane, its grayish color, the difficulty of detaching it, its tendency to extend beyond the limits of the tonsils and the small amount of redness and swelling of the surrounding mucous membrane. If there is also involvement of the nose or larynx the case is almost certainly diphtheria. Membranous inflammation in a child is much more likely to be diphtheria than in an adult. A BACTERIOLOGICAL EXAMINATION should always be made when possible. For this purpose a sterile swab is first rubbed over the suspicious areas in the throat and then over the surface of a tube or plate of Löffler's blood-serum. This is then kept at 100° F. for 12 or 15 hours and smears from the visible colonies are made, stained in the usual way and examined microscopically. A diagnosis can sometimes be made from stained smears made directly from the throat, but the cultural method is much more certain and satisfactory.

Prognosis.—The mortality ranges from 10 to 20 per cent. It varies enormously with the age of the patient, the character of the epidemic, the location of the membrane, etc. The disease is most fatal in young children; in laryngeal cases, in cases of septic diphtheria, and in those in which the toxic symptoms are pronounced. Every case of diphtheria is serious and the prognosis should always be guarded, since in even the mildest cases there exists the possibility of sudden heart failure or other grave complications. Since the introduction of the antitoxin treatment the mortality, in cases in which it has been used early, has been reduced to less than 5 per cent.

Prophylaxis.—Since the infectious material is spread only through the discharges of the mouth and nose, the prevention of contagion is not difficult if only intelligent care is exercised. Every suspicious case should be isolated at once until the diagnosis is made certain. Since it is the mild and unrecognized cases that are responsible for much of the spread of the disease rigid isolation and disinfection should be carried out as described under Scarlet Fever. The bacilli often remain in the throat for some days, or even for several weeks, after the disappearance of the membrane, and during this time there is danger of conveying the disease. Throat cultures, when possible, should be made from time to time and the patient kept in quarantine as long as they show the bacilli to be present. If such bacteriological examinations are impossible quarantine should be maintained in mild cases for at least ten days, in severe cases for at least three weeks, after the disappearance of the membrane. The well children in the infected household must be kept from school and from mixing with other children.

Treatment.—General Management.—All cases, even the mildest, should be kept in bed until convalescence is well established. In the sick-room fresh air, free ventilation and an equable temperature are essential. The diet should be fluid—milk, broths, whey, etc.—and should be given at two-hour intervals. Water should be freely given. Care must be taken that the child be not too frequently disturbed by the various therapeutic measures employed.

Drugs.—It is doubtful if any drug known has any direct, favorable influence upon the course of the disease. The tincture of the chloride of iron and the preparations of mercury which formerly were much used have been largely given up. Stimulants are needed in most of the cases during the latter part of the disease and should be administered freely when the heart shows signs of weakness. Alcohol and strychnine are the drugs chiefly to be relied upon. In sudden heart failure strychnine subcutaneously is of great value. During convales-
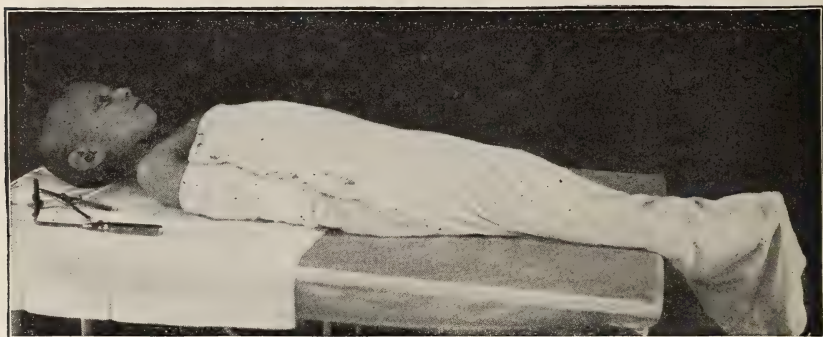
Fig. 70.—Method of wrapping a patient for nasal irrigation or intubation as practiced in the Willard Parker Hospital, New York City. (After Nicoll.)

cence iron, cod liver oil, or other tonics are usually required, as the anemia is apt to be severe.

Local Treatment.—Efforts to destroy the bacilli by strong antiseptic applications are ineffective and often do positive harm. Cleanliness, however, is greatly to be desired. To this end the nose and nasopharynx are freely and frequently irrigated and the mouth and throat sprayed with some bland fluid or mild antiseptic such as normal salt solution, saturated boric acid solution, half strength Dobell's solution, hydrogen peroxide, etc. (See Figs. 70 and 71.) In older children a gargle of very weak (1–10,000) bichloride of mercury may be used. The pain and discomfort of the throat are often mitigated by the use of external applications of heat or cold.

Serum Treatment.—By the brilliant investigations of von Behring, Roux and others a specific method of treatment has now been developed by which the mortality has been greatly reduced and the disease robbed of many of its terrors. This treatment is based upon the fact that by the injection into an animal of repeated and gradually increasing

amounts of the diphtheria toxin there is developed in the blood of the animal a chemical body—*an antitoxin*—which will neutralize the effects of a definite amount of the toxin. In this way animals may be immunized against enormous doses of the diphtheria poison. Moreover, the blood-serum of such immunized animals is capable of neutralizing the effects of the diphtheria toxin in the human body, and also of conferring at least a temporary immunity against the disease to healthy

Fig. 71.—Method of nasal irrigation. The receptacle containing the irrigating solution should be sufficiently above the patient's head to give a gentle flow. (After Nicoll.)

individuals. In practice the blood-serum of highly immunized horses is used after having been sterilized and tested as to its antitoxic strength. This strength is estimated in units, each unit representing 100 times the quantity of serum needed to neutralize the minimal fatal dose of diphtheria toxin for a guinea-pig weighing 250 grammes.

Diphtheria antitoxin is used for two distinct purposes: first, as a CURATIVE MEASURE in those suffering from diphtheria, and second,

to FURNISH IMMUNITY to healthy persons exposed to infection. The curative dose varies from 1000 to 5000 units, according to the age of the patient and the severity of the symptoms. For children over two years of age 2000 or 5000 units is the usual amount. If no improvement is noticed within twelve hours this dose should be repeated. Rarely a third injection may be required. A large, carefully sterilized hypodermic syringe is used and the serum is slowly injected into the subcutaneous tissue of the back, chest or buttock. It is desirable for various reasons to use the most concentrated serum obtainable. The beneficial effects of antitoxin are seen chiefly in cases in which it is used *early* in the disease, *i.e.,* within the first 48 hours. In all severe cases, and even in mild cases in young children, it should be used at once and without waiting for the bacteriological diagnosis. The good effects are usually promptly seen in the lessening of the local redness and swelling, in the loosening of the membrane and in the improvement in pulse, temperature and the constitutional symptoms. This is true of the laryngeal cases as well as of those of pharyngeal diphtheria.

Serious symptoms, as a result of the use of antitoxin, are rare. Skin eruptions, such as erythema or urticaria, together with a rise of temperature and joint pains, occasionally develop from seven to ten days after the injection. They are unpleasant but never serious. No harm results from the use of antitoxin even if the disease proves not to be diphtheria. A few cases of sudden death following the administration of diphtheria antitoxin are on record. Some at least of these deaths have been due to the poisonous effect of the proteid contained in the horse serum (anaphylaxis). Danger from this source is lessened by the use of concentrated sera. Recent studies suggest strongly that asthma is a similar anaphylactic reaction and clinical experience has shown that diphtheria antitoxin is especially dangerous to asthmatics. Antitoxin should therefore not be given to asthmatic patients. Since a latent asthmatic tendency may well exist in a child without the knowledge of either parents or physician, it is advised in all cases to test the susceptibility of the patient by the injection of a minute quantity of the serum (0.5 c.c.) and to follow this by the full curative dose in ten or fifteen minutes if no reaction develops. Susceptible individuals react almost instantly to even the minute quantity named.

Immunizing doses of from 300 to 500 units should be given at once to all children who have been exposed to infection. By such a dose practically complete immunity is secured for a period of at least several weeks. Nurses or other adults exposed to infection may be protected in like manner.

TREATMENT OF LARYNGEAL CASES.—Every case of primary membranous laryngitis should receive a prompt and liberal dose of antitoxin (5000 units or more), for in no type of diphtheria are the results more gratifying, and the dose repeated in 12 hours if necessary. The air that the child breathes should be warmed and moistened by the use of a steam croup kettle near the bed. Calomel fumigations are recommended. A

tent is made over the child's crib and filled with the fumes of calomel and these are inhaled for 15 or 20 minutes at intervals of from one to three hours. Ten or fifteen grains of calomel are placed in a pan or on a metal sheet and heated by an alcohol lamp placed beneath, great care being taken to avoid fire. In the intervals between the fumigations the tent should be removed and the room aired. If the stenosis is progressive and becomes serious, intubation or tracheotomy should be resorted to. Of late years the former has replaced tracheotomy and is now the measure usually employed.

Intubation.—The INDICATIONS for intubation are such a degree of dyspnea from laryngeal stenosis as to cause definite cyanosis with retrac-
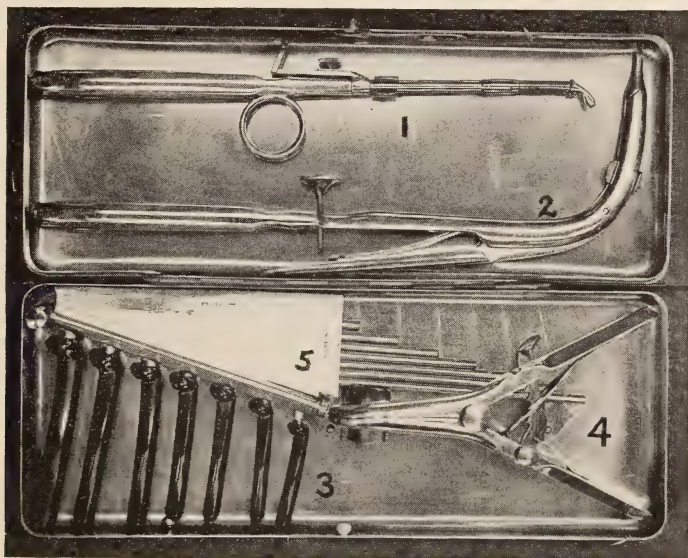
FIG. 72.—The O'Dwyer intubation set. 1. The introducer. 2. The extractor, the tip turned up instead of down. 3. The set of hard-rubber tubes, adapted to children from 1 to 12 years of age. 4. The mouth-gag. 5. The scale to determine the age for which each tube is adapted.

tion of the soft tissues in the episternal notch, the supraclavicular spaces, and the epigastrium. In some instances care is required to distinguish laryngeal stenosis from the cyanosis and dyspnea occasioned by bronchopneumonia.

The INSTRUMENTS and procedure are those devised by Joseph O'Dwyer (see Fig. 72). The child may be held in either of the two positions shown in Figs. 73 and 74. Care is especially necessary to have the head and neck in a straight line. The child being in the proper position, the tube indicated by its age and size is selected, a silk thread run through the "eye" of the tube and the tube secured by its metal obturator on the handle of the introducer. Taking the introducer in the right hand and sitting or standing as indicated in the illustrations,

the operator introduces the left index finger till its tip rests upon the arytenoid cartilages and holds the epiglottis flat upon the base of the tongue.   The tip of the tube is then passed along the anterior surface of the finger as a guide until it reaches the upper aperture of the

Fig. 73.—Intubation in the sitting position.   Note the manner of holding the patient, steadying the head and controlling the mouth-gag.   (After Nicoll.)

larynx.   It is then thrust home, *i.e.*, into the larynx, by raising the right hand and pressing the introducer flat on the tongue, while at the same time the left index finger is slipped onto the head of the tube, and presses it firmly into the larynx, while the introducer and the obturator of the tube are withdrawn, leaving the tube in the larynx.

The tube is often coughed out in the course of a day or two and not required again. If stenosis returns the tube must be replaced. In a few cases repeated introductions must be made.

If the tube remains in the larynx EXTRACTION is practiced after two or three days.

In this procedure the positions of patient and operator are the same as in the introduction of the tube. The left index finger is passed down upon the arytenoid cartilages and the high rear edge of the tube. The tip of the extractor is then made to follow the finger into the lumen of the tube. When the tip engages in the lumen the pressure of the

FIG. 74.—Intubation in the supine position. Note again the manner of steadying the head and controlling the mouth-gag. (After Nicoll.)

thumb upon the lever of the extractor opens the blades of the bill of the instrument and clutches the tube, which is then withdrawn by reversing the movement of intubation.

In infants without molar teeth both these procedures may be practiced without a mouth-gag, but in other children the gag is necessary. In infants after the introduction of the tube the thread running through the eye may be carried into the angle of the mouth secured on the cheek by a bit of adhesive plaster, and later used to extract the tube.

Older children are likely to pull out the tube by the thread and it must therefore be cut and withdrawn after the introduction, the

primary purpose of it being to serve for withdrawal of the tube in case of unsuccessful attempts to place the tube in the larynx. O'Dwyer always insisted upon the necessity of previous training on the cadaver before attempting intubation in the living and few indeed can succeed without such training.

The early use of antitoxin has almost done away with the need of either intubation or tracheotomy, so that intubation is in this country now a rare procedure and tracheotomy for the stenosis of diphtheria is almost unheard of.

FEEDING INTUBATED PATIENTS.—The tube interferes with the closure of the larynx by the epiglottis and liquids often enter the larynx and give rise to severe coughing. To avoid this semi-solid foods should be used rather than liquids, and if the difficulty is pronounced, the Cassel-bury position should be tried. In this the child is laid upon the back upon the nurse's lap, the head being allowed to fall over one side, and the child is fed in this position either from a spoon or through a tube.

DIPHTHEROID INFLAMMATIONS

(Pseudo-Diphtheria)

Definition.—The term DIPHTHEROID is applied to those pseudo-membranous inflammations of the throat and upper air passages which are excited by bacteria other than the Klebs-Löffler bacillus. The STREPTO-COCCUS PYOGENES is the germ usually dominant. It is often associated with the *staphylococcus aureus* or *albus*. Such inflammations are by no means uncommon, and occur in both a primary and a secondary form.

The PRIMARY CASES begin usually as a tonsillitis. A false membrane soon appears, however, which may be indistinguishable from that of true diphtheria. Often it is of a yellowish color and more friable and less firmly adherent than that of diphtheria. It shows also much less tendency to extend beyond the limits of the tonsils. The redness and swelling of the throat, however, are usually more pronounced than in true diphtheria. Clinically the cases show a more abrupt onset, a higher temperature and a shorter course than diphtheria. These primary cases are rarely fatal—the mortality being not more than two or three per cent.

The SECONDARY TYPE is seen as a complication of such diseases as scarlet fever, measles, typhoid, etc. True diphtheria itself may be associated with such diseases, but this happens only infrequently and then usually during convalescence rather than in the active stage of the primary disease. While some of the cases of the secondary type may be mild the form is as a rule severe. The membrane is often extensive, may involve the nose or larynx and is often associated with sloughing and deep suppuration, as is well illustrated in the anginose type of scarlet fever. In many cases a condition of general septicemia or pyemia develops. This secondary type differs greatly in its prognosis

from the primary one. Most cases are very severe and prolonged and as many as one-third or one-half of them prove fatal.

Diphtheroid inflammations seem to be slightly, if at all, contagious and so do not require the rigid and long isolation necessary in diphtheria. Nevertheless, such cases should be kept apart from other children and the discharges from the throat and nose disinfected.

Local treatment should be along the lines suggested for diphtheria. The use of antistreptococcus serum has hitherto not been satisfactory.

WHOOPING-COUGH

(Pertussis)

Definition.—Whooping-cough is a contagious disease of epidemic character occurring in childhood, whose chief feature consists in severe paroxysms of coughing which terminate in a shrill inspiratory " whoop " and are often followed by vomiting.

Etiology.—The disease is one of the common affections of childhood and is distributed widely over the globe. Although met with sporadically, it occurs chiefly in epidemics which are commonest in the winter and spring and are frequently associated with epidemics of measles. Most of the cases are found in children under ten years of age. Young infants are often attacked, as are sometimes also adults. It is commoner in girls than in boys. The disease is highly contagious throughout its whole course. It is contracted usually through close contact, but may be conveyed by fomites or even by means of a third person. The exciting cause is still unknown. Several micro-organisms have been found in the sputum, but none has yet been proven to be the real cause of the disease. Second attacks are not common.

Morbid Anatomy.—No characteristic lesions are found after death. There is probably always a catarrhal inflammation of the larynx, trachea and larger bronchi, which, however, does not explain the peculiar paroxysmal character of the symptoms. The tracheal and bronchial lymph-glands are often found enlarged and the fatal cases show such complications as bronchopneumonia, vesicular or interstitial emphysema, and meningeal congestion and hemorrhage. Miliary tuberculosis is also a frequent finding in fatal cases.

Symptoms.—THE PERIOD OF INCUBATION is commonly of about ten days' duration. Only rarely is it less than seven or more than fourteen.

The symptoms are conveniently divided into three stages: the catarrhal, the paroxysmal, and the stage of decline.

CATARRHAL STAGE.—The onset is gradual and insidious with the symptoms of a mild bronchitis or "cold." There are slight fever, irritability, coryza, and a dry cough which is not paroxysmal. Examination of the chest shows nothing more than an occasional bronchial or moist râle.

PAROXYSMAL STAGE.—In the course of a week or ten days the cough

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becomes more spasmodic and the characteristic whoop appears. The paroxysms consist of a series (ten to twenty) of short, hard coughs coming in rapid succession and ending in a long, deep inspiration which has a crowing or whooping sound. These coughs and whoops may be repeated several times and the paroxysm ends by the child raising a plug of tenacious mucus from the larynx or trachea and often by vomiting as well. During the coughing there are great congestion of the head and neck, enlargement of the veins, suffusion of the eyes and often marked cyanosis, all of which are relieved after the crowing inspiration. Such paroxysms vary in number from five or six to forty or fifty in the twenty-four hours. These attacks may be excited by eating, drinking, crying, etc., but are often spontaneous and are usually especially frequent during the night. The constant vomiting seriously interferes with nutrition and the child grows thin, pale and listless. The temperature, as a rule, remains normal and the chest presents no characteristic physical signs. The blood regularly shows a marked leukocytosis with an increase in the proportion of lymphocytes. This stage usually lasts from two to four weeks, but in severe cases may be much longer. Occasionally the paroxysms are indistinct and slight and the whoop may be lacking altogether.

STAGE OF DECLINE.—The spasms of coughing grow less frequent and severe and finally cease, although some cough persists for weeks. This stage usually lasts from three to four weeks and the whole period of the disease is commonly from eight to twelve weeks, but may be much longer.

Complications.—Bronchopneumonia is the most common and serious complication, especially in young children. It may be tuberculous. Vesicular emphysema is frequent. Rupture of air vesicles may result in extensive infiltration of the tissue with air. Hemorrhages, from the violent coughing efforts, are common in the nose and conjunctiva and may occur in the bronchi, ears, meninges, etc. Convulsions are met with in young children and may be fatal. Hemiplegia, aphasia, disturbances of sight, etc., occur rarely. The persistent vomiting may result in great anemia and emaciation. Diarrhea is not uncommon.

Diagnosis.—This rests upon the history of exposure, the severe spells of coughing, the peculiar whoops, the long-continued cough without fever or physical signs in the lungs. In mild and doubtful cases a leukocytosis with a marked lymphocytosis would point strongly toward pertussis. In a child an acute bronchitis which fails to improve under rational treatment should always be regarded with suspicion.

Prognosis.—The disease is very fatal in infants, chiefly from bronchopneumonia or convulsions. In older children among the better classes, the mortality is slight, but the patients are often long in recovering from the exhausting effects of the disease. Tuberculosis is not infrequently a sequel.

Treatment.—Patients should be carefully kept away from other children and from school throughout the disease, but should not be con-

fined to a room. Fresh air is of the greatest importance and they should be kept out of doors as much as possible. The feeding demands much attention, because of the great tendency to vomiting, and everything should be done to maintain the general health. Plain, nutritious food should be given in small quantities, and at short intervals.

No method of treatment is known by which the disease can be cut short. All that can be hoped for from drugs is to diminish the frequency and severity of the paroxysms. No one drug seems to act best in all cases. The following list includes some of the more important drugs with the average *daily* amount needed for a child of two years: Quinine gr. vi, antipyrine gr. viii, tincture of belladonna m. xv, sodium bromide gr. xx, chloral gr. x, bromoform m. v.

Inhalation of creosote, carbolic acid, formaldehyde, cresolin, etc., is sometimes useful, and a change of air is often of benefit.

MUMPS

(Epidemic Parotitis)

Definition.—A specific contagious disease characterized by inflammation of the parotid glands and a tendency to metastatic inflammation of the sexual organs.

Etiology.—Mumps is a disease chiefly of older children, but is occasionally seen in infancy and in adult life. It appears usually in epidemic form and during the winter months. Contagion occurs, as a rule, only after close contact. Transmission through a third person or by fomites is rare. The nature of the contagium is unknown. Boys seem rather more susceptible than girls.

Morbid Anatomy.—The lesion is an acute exudative inflammation of the parotid gland and its surrounding connective tissue, which terminates regularly in resolution. Suppuration is rare and accidental. Occasionally the other salivary glands may be involved.

Symptoms.—The INCUBATION PERIOD is quite regularly from two to three weeks. In mild cases *pain* is often the first symptom. In the severer form the local symptoms are usually preceded by a day or two of malaise, joint pains, headache and fever.

The fever in such cases may reach 103° or 104° F., and lasts for several days. In mild cases it is very slight. The local symptoms begin as a rule with pain in the parotid region, followed promptly by swelling of the gland, which in two or three days reaches its height. The swelling is located chiefly below and in front of the ear and when marked, as it often is, displaces the lobe of the ear and gives to the face a singular and ludicrous expression (see Fig. 75). The swelling is boggy and tender, but not red. Both glands are commonly affected; the signs in the second one appearing usually a day or two after those of the first. The pain is felt in the glands, in the throat, and often in the ears, and is made worse by swallowing and by the presence of tart

substances in the mouth. Often the jaws cannot be widely opened. The saliva is frequently much diminished. The swelling subsides in from five to ten days, while the constitutional symptoms are usually of much shorter duration.

Complications.—In males beyond the time of puberty orchitis is a common complication. It may affect one or both testicles. It appears late in the disease, lasts four or five days and ends in resolution. It may be followed by atrophy of the testicle. In girls inflammation of the vulva, the breasts and the ovaries sometimes occurs. Rarely cerebral symptoms such as delirium and convulsions appear. Meningitis, deafness, nephritis, multiple neuritis and endocarditis are possible complications.
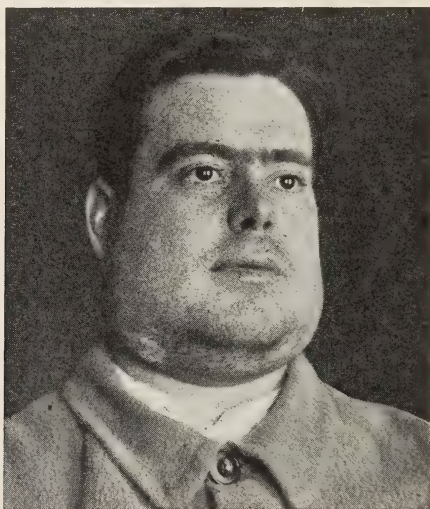
Fig. 75.—Mumps: Note how the swelling extends upward in front of the lobe of the ear.

Diagnosis.—The disease is usually easily recognized. It is to be distinguished from the secondary form of parotitis, which is usually suppurative. Occasionally it may be confused with swelling of the cervical lymph-nodes.

Prognosis.—Mumps regularly terminates favorably and usually runs a mild and short course. Rarely death may occur from some complication.

Treatment.—Patients should be kept away from other children and from school for at least a week after the swelling has entirely subsided. Rest in bed is needed during the febrile stage. If there be much pain moist heat applied to the gland is usually comforting. Fluid diet or soft foods should be used when there is much pain and difficulty in swallowing.

TABLE OF COMPARISON OF THE COMMON ACUTE CONTAGIOUS DISEASES

Disease	Incubation	Invasion	Appearance of eruption	Early location of eruption	Desquamation	Period of quarantine
Measles............	10 to 14 days	Coryza	4th day	Behind ears and on neck and face	Branny	2 to 3 weeks, or till skin is clear
Scarlet fever........	1 to 7 days, usually 4	Fever, headache, sore throat and vomiting	12 to 48 hours	Chest, arms and legs inner aspects	Strips or sheets	5 to 6 weeks, or till skin is clear
German measles......	2 to 3 weeks	Fever, malaise, rarely severe	At once	General	None or branny	Two weeks, or till skin is clear
Chicken-pox.........	2 to 3 weeks	Mild	At once	Back or face	Thin crusts	Until skin is clear, 1 to 2 weeks
Small-pox...........	10 to 12 days	Severe fever, headache, back-ache	3d to 4th day	Face	Heavy crusts	Until skin is clear, 2 to 3 weeks
Whooping-cough......	About 2 weeks	Increasing cough	....	....	....	Until whoop ceases, usually 6 weeks
Mumps..............	2 to 3 weeks	Pain and swelling of parotid	....	....	....	Three weeks
Diphtheria..........	1 to 7 days, usually 2 to 4	Sore throat, fever, depression	....	....	....	Until bacilli disappear from throat (2 cultures)

SEPTICEMIA AND PYEMIA
(Blood Poisoning)

Definition.—Septic diseases are those caused by the invasion of the blood and tissues generally, by pyogenic bacteria or their toxic products. Chief among these pus-forming agents are the *streptococcus pyogenes* and *staphylococcus pyogenes aureus*.

Three types of septic disease are recognized: (1) SEPTIC INTOXICATION (SAPREMIA); in which the bacteria are confined to the local lesion and in which the general symptoms are caused altogether by the absorption and dissemination of the bacterial toxins.

(2) *Septicemia (Bacteremia)*; in which the bacteria themselves gain entrance to and multiply in the blood and tissues. The term septicemia is also sometimes used to include septic intoxication.

(3) *Pyemia* or *Septico-pyemia;* in which in addition to a septicemia there is the development of metastatic abscesses.

SEPTIC INTOXICATION

The constitutional disturbance in every form of local purulent inflammation, in which the causative bacteria are not disseminated throughout the body, is an instance of septic intoxication. Empyema, purulent otitis media, appendicitis, erysipelas and certain forms of puerperal infection are familiar examples. The severity of the symptoms depends upon the nature of the toxin, the amount produced and especially upon the rate of absorption. The symptoms are chiefly fever, headache, thirst, anorexia, prostration, rapid pulse and, usually, leukocytosis. Practically it is often very difficult to distinguish between the severer forms of septic intoxication and a true septicemia or bacteremia. In the former case the symptoms should subside promptly upon the subsidence or removal of the local inflammatory process. When, however, the bacteria have entered the general circulation no such close relation is seen between the constitutional symptoms and the state of the local infection.

SEPTICEMIA

Etiology.—Any local infection, however small and apparently insignificant, may be the starting point of a septicemia. Among the more important of such causes are puerperal infection of the uterus, acute osteomyelitis, acute otitis media with its complications of mastoid inflammation and septic thrombosis of the lateral sinus, post-mortem and other infected wounds, suppurative appendicitis, gonorrhea, liver abscess, etc. A very important class of septicemias is that in which during life (and even sometimes after death) the point of entrance of the bacteria cannot be determined—*idiopathic* or *cryptogenetic septicemia*. Such cases are by no means rare and come under the eye of the physician rather than the surgeon. They are prone to develop in those whose vital forces and powers of resistance are weakened by prolonged ill-

ness, and are frequently seen in the last days of many chronic diseases and cachexias.

BACTERIA.—Of the exciting causes of septicemia the streptococcus pyogenes and the staphylococcus aureus and albus are much the most common. Other organisms less frequently met with are the pneumococcus, gonococcus, bacillus coli communis, bacillus pyocyaneus, bacillus aërogenes capsulatus, etc. The bacteria may gain entrance to the circulation by way of the lymphatics or by direct infection of the small veins in the vicinity of the primary focus either with or without an accompanying septic thrombosis.

Morbid Anatomy.—The lesions found at autopsy are not peculiar to the disease. The blood is fluid and very dark, the viscera are congested and show parenchymatous degeneration, the spleen is swollen and soft, and hemorrhages into the serous membranes are common.

Symptoms.—These appear usually within a very few days after those
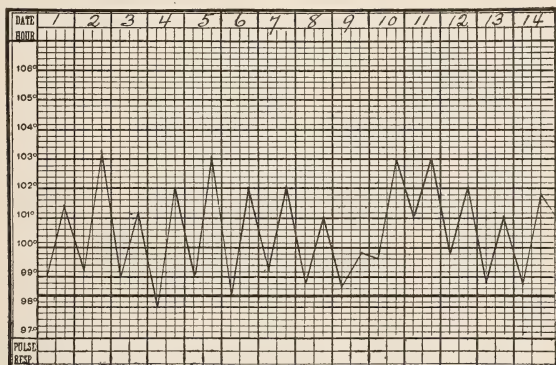
Fig. 76.—Temperature curve of septico-pyemia.

of the local infection. The symptoms are often ushered in by a severe rigor and sweat, which may be repeated at irregular intervals throughout the illness. The fever is high and very irregular, often rising to 105° or 106° F., and dropping suddenly to normal or below. (See Fig. 76.) There are marked and increasing prostration, a very rapid, small, soft pulse and a dry tongue. The mind may remain clear, but if the symptoms last for many days a " typhoid state " usually develops with muttering delirium or dulness and apathy. A severe diarrhea is common. The skin assumes a peculiar yellowish pallor or there may be distinct jaundice. Hemorrhages are frequently seen in the skin and mucous membranes. The blood shows a rapidly progressive anemia and, in almost all cases, a marked polymorphonuclear *leukocytosis.* By proper methods the bacteria can usually be demonstrated in the circulating blood.

The *course* varies much. The severest cases are fatal within two or three days. Most of the cases last from five to ten days and not a few

of these are fatal. In those cases which recover convalescence is apt to be slow and tedious. Most of the very acute and severe forms of septicemia are caused by the streptococcus. Malignant endocarditis is a not very uncommon complication.

Diagnosis.—If a cause for the general infection can be found, such as a septic uterus or a post-mortem wound, the diagnosis offers no difficulty. In the cryptogenetic cases the symptoms of chief diagnostic importance are the irregular temperature, the chills and sweats, the rapid prostration, the purpuric spots, the high leukocytosis and the presence of pyogenic bacteria in the blood. The conditions most likely to be confused with septicemia are malarial fever, the intermittent hepatic fever caused by impaction of a gall-stone in the common duct, malignant endocarditis, typhoid fever and certain types of pulmonary tuberculosis.

Treatment.—The local infection should receive prompt and radical surgical treatment. The patient's strength should be maintained as well as possible by nutritious and easily digested food and by stimulants. The bacterial toxins should be diluted and their rapid elimination encouraged by high rectal irrigations or subcutaneous or intravenous infusions of normal salt solution, and by the free drinking of water. If the infectious agent is known to be the streptococcus pyogenes, anti-streptococcic serum may be tried. Or if the causative bacteria can be found in the blood, vaccines made after Wright's method may be employed.

PYEMIA

A certain proportion of the cases of bacterial invasion of the blood and tissues (septicemia) is associated with the formation of multiple abscesses in the viscera and other structures. To these cases the name pyemia or septicopyemia is given.

The ETIOLOGY of pyemia does not differ from that of septicemia in general. The sources of infection and the varieties of bacteria are the same. Metastatic abscesses are especially apt to develop, however, when the local infection sets up a septic thrombo-phlebitis in the neighboring small veins. Fragments of the friable thrombus, containing the bacteria, are readily detached and may be carried by the blood stream to distant parts of the body and there excite local suppuration. Usually the septic emboli are carried first to the lungs, and these organs are therefore a favorable site for the secondary abscesses; but often minute particles pass through the lung capillaries, enter the arterial system and may there produce septic infarcts and abscesses in the spleen, kidneys, brain, joints, muscles, subcutaneous tissue, etc. Septic infections of the endocardium may occur and from this malignant endocarditis other septic emboli may result. When the primary local infection is in the region drained by the portal vein (*e.g.*, in the appendix) the emboli lodge chiefly in the liver and there set up multiple abscesses. The SYMPTOMS of pyemia are those of septicemia plus those of the metastatic abscesses.

Chills and sweats are even more common than in septicemia alone and often show a striking periodicity, so that they may resemble closely those of intermittent malarial fever. The *course* of the disease is usually not so rapid as that of the most virulent type of septicemia and most of the cases last from one to three weeks. In some of the milder forms the course is still more chronic and the conditions may persist for several months. The PROGNOSIS in most cases is bad, although many of the chronic cases (usually staphylococcic infections) eventually recover.

The DIAGNOSIS is made by the recognition of a septicemia and the appearance of metastatic abscesses.

The TREATMENT of pyemia does not differ from that of septicemia, except that the abscesses, when they appear, demand surgical treatment.

ERYSIPELAS

Definition.—Erysipelas is an acute contagious inflammation of the skin caused by the *streptococcus erysipelatis,* an organism not distinguishable from the streptococcus pyogenes.

Etiology.—The disease is wide-spread and occurs endemically, sporadically, and occasionally in small epidemics. It is commonest during the spring months and is seen at all ages and in both sexes. Chronic alcoholism, debilitating chronic diseases, unhygienic surroundings and uncleanly habits are predisposing causes. Liability to the disease is much increased by the presence of open wounds and by the puerperal state. Under modern aseptic methods erysipelas has now become rare in the surgical wards where once it was a scourge. The streptococcus erysipelatis seems to be very closely related to, if not identical with, the streptococcus pyogenes. The cocci are found in great numbers in the lymphatics of the inflamed skin especially at and just beyond the spreading margin. Infection occurs probably always by inoculation of a wound or an abraded skin or mucous surface, although frequently the point of infection cannot be discovered. The germs may be carried by the hands of physicians and nurses, by instruments, dressings, fomites and, apparently, also by the air. Often it is impossible to trace the origin of the disease.

Morbid Anatomy.—The process is a severe acute inflammation of the entire thickness of the skin, and often of the subcutaneous areolar tissue, with intense hyperemia and marked infiltration of the tissues with serum, leukocytes and fibrin. In severe cases cutaneous abscesses may develop.

Symptoms.—The INCUBATION PERIOD lasts from three to seven days. The ONSET is abrupt and sometimes violent. An initial *chill* is common; there is frequently vomiting and the temperature rises rapidly to 103°– 105° F. This is associated with headache, fever, pains and a rapid, bounding pulse.

LOCAL SYMPTOMS.—In TRAUMATIC erysipelas the inflammation shows itself usually in the vicinity of the infected wound. In the IDIOPATHIC

form the process, in most cases, appears upon the head or face, although it may begin at any part of the body. Within a few hours of the onset, in a typical case, a small area of skin on the cheek or over the bridge of the nose becomes red and swollen and this spot rapidly increases in size so that within twenty-four hours a considerable area of the face is involved. The affected skin is bright red, swollen, hot, shining and indurated. The advancing edge of the inflammation is sharply defined from the normal skin and is distinctly elevated. The process spreads rapidly to the forehead, ears, lips and often to the hairy scalp. The face is enormously swollen, the eyes are closed by subcutaneous edema, and the features are often quite unrecognizable. The process sometimes extends to the mucous membrane of the nose, mouth or throat. Frequently serum collects beneath the epidermis in small blisters or larger blebs and in severe cases there may be small cutaneous abscesses. In most cases of facial erysipelas the inflammation stops at the neck. A
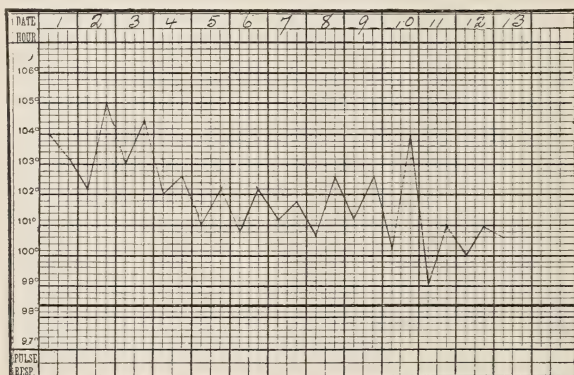
FIG. 77.—Temperature curve of erysipelas of moderate severity.

migratory type is sometimes seen, however, in which it slowly extends from place to place until the greater part of the body has been covered. The inflammation lasts usually from five to ten days. During this time the fever persists, but falls rapidly to normal as the local process subsides. (See Fig. 77.) Albuminuria is common and there may be a troublesome diarrhea. In severe or protracted cases delirium or the so-called '' typhoid state '' may develop.

Complications.—A number of severe complications, such as pneumonia, nephritis, edema of the glottis, malignant endocarditis, septicemia, pyemia and meningitis, are possible, but fortunately none of them is common.

Diagnosis.—The marked constitutional disturbance and the characteristic local appearances (the bright red, swollen and indurated skin with its sharp, elevated margin) are the features of special diagnostic importance.

Prognosis.—The disease is rarely fatal except in those suffering from severe surgical affections or in debilitated or alcoholic subjects. A form of the disease occurring in new-born infants from infection at the umbilicus is very fatal.

Treatment.—Isolation and disinfection are required as in the case of other contagious diseases.

The constitutional symptoms are to be treated symptomatically. The patient's strength should be maintained by nutritious and easily digestible food and when necessary by stimulants such as alcohol or strychnine. Tr. ferri chloridi (m. xx–xl t.i.d.) has been much used, but its efficacy is doubtful.

LOCAL TREATMENT.—Ichthyol, in the form of 10 to 30 per cent. ointment, is perhaps the most satisfactory. Compresses soaked with saturated boric acid solution or a weak carbolic solution (1–50) or with lead and opium wash often give much relief. Injections of carbolic solution (1–50) into the skin just beyond the inflamed margin have sometimes succeeded in limiting the inflammation. In many cases, however, the disease runs its self-limited course apparently uninfluenced by any method of treatment. In some cases repeated injections of antistreptococcic serum appear to have been beneficial and in a few instances have seemingly stopped the progress of the disease.

ACUTE RHEUMATIC FEVER

(Acute Articular Rheumatism)

Definition.—An acute febrile disease, probably of infectious nature, characterized by multiple arthritis and a tendency to inflammation of the heart and of various fibrous tissues.

Etiology.—PREDISPOSING CAUSES.—Acute rheumatism prevails chiefly in temperate and moist climates. In this country most cases are seen during the late winter and spring months. In England, however, it is most frequent in the autumn. It occurs for the most part endemically, but occasionally appears in small epidemics.

AGE.—The disease is seen chiefly in adolescence and young adult life; the great majority of cases occurring between the ages of fifteen and forty years. It is by no means infrequent in children between five and fifteen years, but is rare in infancy and in old age.

SEX.—In children rheumatism is commoner in girls than in boys, but in adult life males are the more frequently attacked.

HEREDITY plays a less important rôle than is popularly supposed, although a certain predisposition seems occasionally to be transmitted. The continued *exposure to cold and damp*, and to sudden changes of temperature, distinctly predisposes to the disease and it is frequently met with among coachmen, truckmen, bakers, laborers, etc. One attack does not provide immunity, but rather increases the liability to the disease.

Bacteriology.—Various micro-organisms, in particular a micrococcus

resembling in many ways the streptococcus pyogenes, have been suggested as the cause of the disease, but for all of these satisfactory proofs are still lacking. Poynton and Paine have especially studied the problem and have isolated from the local lesions an organism which they have called streptococcus rheumaticus and regard as the specific agent. The belief that rheumatism is an infectious disease is, however, almost universal. It has many points of similarity with septicemia and is looked upon by some as a modified form of that disease. The tonsils are now regarded as the probable portals of entry for the infectious agent. By some it is held that the symptoms are not caused by a single specific germ but may be produced, under certain conditions, by all the common pyogenic organisms. The older views that the disease was due to the accumulation in the tissues of lactic or uric acids (chemical theory), or to a primary disturbance of the nerve-centers controlling the nutrition of the joints (nervous theory), have been almost wholly abandoned.

Morbid Anatomy.—The lesion of the joints is an acute exudative inflammation affecting both the synovial membrane and the peri-articular structures, the exudate consisting of serum and some fibrin. Only very rarely is the exudate purulent. From its character the inflammation is capable of, and usually undergoes, complete resolution, and permanent damage to the joints is very unusual. The most frequent lesion in the heart is an acute, simple endocarditis, leading usually to sclerosis and deformity of the valves. Acute pleurisy or pericarditis (fibrinous or serous) and acute myocarditis are much less common.

Symptoms.—In many cases there are for a day or two slight PRODROMATA such as malaise, sore throat and vague joint pains. Often, however, the ONSET is abrupt, with pain and swelling of one or two joints and a rapid rise of temperature. A distinct chill is uncommon. Within 24 or 36 hours the disease is fully developed. The AFFECTED JOINTS are extremely painful, especially upon the slightest movement, and are swollen, somewhat reddened and exquisitely tender. There is usually only a moderate quantity of fluid in the joint cavity, much of the swelling being due to the inflammatory edema of the peri-articular tissues. The knees, ankles, elbows, wrists and shoulders are the joints chiefly affected. Less frequently the phalangeal joints are inflamed, as are rarely also the vertebral, sterno-clavicular and temporomaxillary articulations.

The inflammation regularly attacks several joints; and it is a very characteristic feature of the disease that these are involved somewhat in succession, the swelling and pain suddenly leaving one joint and appearing in another.

THE FEVER is usually only moderately high (102°–103° F.), is irregular and is commonly associated with profuse sweating; the sweat being acid in reaction and having a sour, pungent odor. Sweat vesicles (sudamina) frequently appear over the skin of the trunk. The pulse is moderately quickened, the tongue moist and heavily coated, the bowels constipated and the urine scanty, high-colored, and very acid. The blood shows a

very rapidly progressive secondary anemia and usually a distinct leu-kocytosis. The symptoms commonly last from ten days to two weeks, but recrudescences and relapses are very common and it is not rare to have the course, even when uncomplicated, protracted to five or six weeks. To some of these cases with a mild but very protracted course the term *subacute rheumatism* may properly be applied.

Symptoms in Children.—The joint symptoms are usually much less conspicuous than in adults and may appear only in the form of "growing pains"; or they may pass altogether unnoticed. On the other hand, the cardiac manifestations are frequent and serious. They may appear before the joint symptoms or they may be the only evidences of the disease. The secondary anemia is often severe.

Complications.—THE HEART.—Acute endocarditis is the commonest heart complication. It occurs in from one-fourth to one-half of the cases. It is especially frequent in children and in those who have had several attacks of rheumatism. It is regularly of the simple type—ulcerative endocarditis being exceedingly rare. The mitral cusps are much more frequently attacked than are the aortic. The condition may manifest itself by some increase in the temperature and pulse rate and by some precordial discomfort, but very often it gives no symptoms by which it can be recognized. The physical signs also may be very mis-leading. A systolic murmur over the apex or base of the heart is by no means proof that an endocarditis exists, since the apical murmur may be caused by a temporary leakage of the mitral valve, due to muscular relaxation, and a murmur over the pulmonic area is often caused by the severe anemia. Often the nature of the murmur can be determined by observing its persistence or disappearance after recovery from the rheumatic attack. Acute endocarditis is in itself usually not a serious matter. Its importance lies in the fact that it is so frequently the starting point of a chronic sclerosing process which results in serious deformity of the valve.

PERICARDITIS is much less common than endocarditis. The exudate may be either fibrinous or serous. It may terminate in recovery or death or may result in obliteration of the pericardial sac and in fibrous thick-ening.

Acute MYOCARDITIS is occasionally met with. There is granular and fatty degeneration of the heart muscle, together with more or less exuda-tion of serum and leukocytes into the interstices. It frequently results in dilatation and in serious weakening of the heart action.

HYPERPYREXIA.—This is a grave but fortunately very rare compli-cation. It is apt to develop in the latter part of the attack and without apparent cause. The temperature may rise rapidly to 106° or 108° F., is usually associated with delirium and cardiac weakness and often ter-minates in coma and death. Delirium occasionally occurs without hyper-pyrexia. It may then result from the rheumatic poison itself or may be due to a complicating pericarditis or to the toxic effects of the salicy-lates.

Skin Affections.—The frequency of sudamina has been mentioned. Erythema nodosum and erythema multiforme, urticaria and purpura are all occasionally met with.

Rheumatic Nodules.—In children, and especially in those who have had several rheumatic attacks, there frequently develop subcutaneous fibrous nodules, varying in size from a small shot to a bean. They are usually seen over the extensor surface of the elbows, forearms and wrists, over the patella and shins and along the vertebral spines. As a rule they represent a severe type of rheumatic infection. They are much more common in England than in this country.

Chorea.—Chorea is sometimes, though by no means always, a rheumatic manifestation. It may appear before, with or after the joint symptoms.

Lobar pneumonia and pleurisy are occasional complications. Acute tonsillitis and pharyngitis are very frequently present at the onset, and may occur at any time during the attack.

Diagnosis.—The involvement of several large joints, the transient and migratory character of the inflammation, the fever and the sour sweat are the most characteristic features in adults. The history of earlier attacks or the presence of valvular disease or of subcutaneous nodules will lend support to the diagnosis. It is important to remember that in children the joint manifestations are often inconspicuous and slight. A monarticular arthritis is almost never rheumatic.

Gonorrheal arthritis; the acute manifestation of arthritis deformans and gout; scarlatinal ''rheumatism'' and other forms of septic or pyemic arthritis, and acute osteomyelitis are the affections most likely to be confused with rheumatism.

Prognosis.—Rheumatic fever is serious chiefly because of the frequency of the heart complications. The liability to these is much greater in children than in adults and is much increased by each fresh attack of rheumatism. Large and numerous rheumatic nodules are usually of bad omen. Death from hyperpyrexia is rare.

Treatment.—The patient should be put to bed at once and should remain there until convalescence is thoroughly established. Because of the tendency to free sweating it is well to have him wear a flannel night-gown, and lie between blankets rather than sheets. Fluid diet, chiefly of milk, is needed during the febrile stage. Later the diet should be made liberal, foods of all kinds being allowed. Water, lemonade, etc., should be given freely. The inflamed joints should be painted with oil of wintergreen, methyl salicylate or equal parts of guaiacol and glycerine, and enveloped in a thick roll of non-absorbent cotton. Immobilization during the acute stage may help to relieve pain.

Drugs.—Two modes of treatment are in vogue: that with the salicyl compounds and that with alkalies. The salicylates have the power of relieving the pain and lessening the temperature without apparently shortening the course of the disease or diminishing the danger of heart complications. This last the alkalies are believed to do, although they

are much less efficient in relieving pain. The best results are obtained by combining the two methods. Sodium salicylate is the preparation most in use. It should be given in full doses (gr. xv–xxx every three hours) until the pain is relieved or until deafness or ringing in the ears appears, when the doses should be reduced in amount and in frequency. The salicylate is best given by combining with it twice as much bicarbonate of soda. Each dose should be well diluted in plain or carbonated water. If sodium salicylate is not well borne by the stomach salicin (gr. xx–xxx), oil of wintergreen (m. xv–xx) or aspirin (gr. xv–xx) are efficient substitutes.

In the alkaline treatment potassium bicarbonate (gr. xxx) or citrate (gr. xx) is administered every three or four hours until the urine becomes alkaline and is then given in amounts only sufficient to keep the urine neutral or slightly alkaline.

If signs of cardiac mischief appear an ice bag, mustard pastes or blisters may be applied to the precordium and the patient must be confined to bed for a much longer period than usual, indeed until all signs of cardiac disturbance (except the murmurs) have disappeared.

After the fever has subsided, iron, arsenic and the bitter tonics are useful. In the late stages counter-irritation, douching and massage to the joints may aid in hastening convalescence. Hyperpyrexia demands prompt and energetic treatment by cold baths.

GONORRHEAL INFECTION

The gonococcus is capable of exciting a number of disorders other than the common inflammation of the urethra. Some of these, such as epididymitis, prostatic abscess and cystitis in the male, and vaginitis, endometritis, salpingitis and peritonitis in the female, result from direct extension of the process from the urethra. In other instances, however, a general infection results from the absorption of the bacteria and their toxins from the point of primary infection and this may show itself as a virulent septicemia or it may excite local inflammations in various places remote from the primary focus; especially in the joints, tendon sheaths, bursæ, endocardium or pericardium. In every such instance it is probable that the gonococci themselves invade the blood stream, since they have repeatedly been demonstrated in the blood, in fluid from the inflamed joints and in the vegetations upon the heart valves; but it is possible that some of the milder disorders may be caused by the toxins alone.

GONORRHEAL ARTHRITIS

Inflammation of the joints complicates about ten per cent. of the cases of local gonorrheal infection. It is met with in both sexes, though far commoner in males, and has been known to result from gonorrheal ophthalmia in the new-born, and from gonorrheal vulvo-vaginitis in children. The condition manifests itself usually in the third or fourth week

of the urethritis, but is sometimes seen much earlier or may develop in the course of a chronic gleet.

Morbid Anatomy.—The inflammation involves both the synovial membrane and the peri-articular structures (ligaments, tendon sheaths, fascia, bursæ, etc.). The joint is filled with a serous, sero-fibrinous or sero-purulent exudate and the peri-articular tissues are swollen and edematous. Later there may be considerable formation of new connective tissue and subsequent fibrous thickening of the tissues about the joints.

Symptoms.—Distribution.—Any joint may be attacked, but the knees, ankles and wrists are the ones commonly affected. Occasionally the smaller joints (phalangeal, temporomaxillary, sterno-clavicular, intervertebral, etc.) are involved. In nearly half of the cases the inflammation is confined to a single joint (monarticular form), and in many other instances to two joints. The polyarticular form is comparatively infrequent.

The symptoms begin with pain, swelling and tenderness in the affected joint. The fever is usually not high (100°–102° F.), nor are the other constitutional symptoms marked. The joint is moderately swollen, and often only slightly reddened, but the edematous swelling extends for some distance above and below the joint and gives to the whole a fusiform outline. The pain varies greatly in different cases, but it is usually very severe on motion or pressure. The active symptoms run a subacute course and last from two to six weeks, while the stiffness and disability often persist much longer. The tendency of the inflammation to move about from joint to joint, so characteristic of acute rheumatism, is usually quite lacking in this disease. In the polyarticular form the local inflammation is less severe and stiffness and pain on motion may be the chief symptoms. Sometimes the inflammation attacks chiefly the bursæ, tendon sheaths, fasciæ or muscles, with little or no involvement of the joints proper.

Prognosis.—Most cases recover completely, although convalescence is frequently very prolonged and tedious. Occasionally the fibrous ankylosis may be complete and permanent. Death may occur from an associated septicemia or endocarditis.

Diagnosis.—This is not difficult except where no evidence of gonorrhea can be obtained. In such cases a positive diagnosis can be made only by the finding of gonococci in the joint fluid. The subacute course, the involvement of a single joint, the absence of a shifting character to the inflammation, the degree of peri-articular involvement and the ineffectiveness of the salicylates are the chief distinguishing features from acute rheumatism. The condition may be confused with the acute manifestation of arthritis deformans, and with gout, septic or tuberculous arthritis, osteomyelitis, etc.

Treatment.—The salicylates and alkalies seem to have no effect whatever. Tonics and reconstructives, such as iron, arsenic and cod liver oil, are, however, of distinct value. Both antigonococcus serum and

Morbid Anatomy.—Many varieties of brain tumor are known—the common ones are tubercle, sarcoma, glioma, gumma and carcinoma. Tubercle occurs in the form of large, isolated and usually single tumors made up of masses of miliary tubercles or a single large tubercle as much as an inch or an inch and a half in diameter. Tubercle in the form of tumor is common in childhood, rare in later life. Gumma, on the other hand, never results from inherited syphilis, and is therefore very rare in childhood, but common in adult life. Sarcoma or carcinoma may follow a primary growth elsewhere, but is usually primary in the brain. Glioma is a growth of neuroglia, and therefore peculiar to the nervous system. Cystic tumors of the brain may arise from parasitic infections, such as echinococcus or cysticercus. They are very uncommon in this country. Fibroma, angioma, myxoma, osteoma and other rare forms are known.

In brain tumor the intracranial pressure is always increased, and if the skull is opened during life the dura bulges and its pulsation is diminished. The cerebral convolutions are regularly flattened. The ventricles often contain excess of fluid owing to obstruction of the venæ Galeni by pressure.

Symptoms.—These are both general and focal.

GENERAL.—These are produced by the steadily increasing intracranial pressure. (1) Severe and persistent headache. (2) Mental disturbance. The disposition may change notably, and the patient become irritable, careless or indifferent. Memory is impaired and attention fails. The patient may become dirty and untidy in his habits. (3) Vertigo and vomiting. Dizziness is common in any cerebral tumor, but is pronounced if the cerebellum is involved. Vomiting is frequent, and is usually projectile in type. (4) Slow pulse. The rate is slow, 50 to 60, the pulse full and of high tension. (5) Convulsions. These may be general or of the Jacksonian type. (6) Choked disk is present in 80 to 90% of brain tumors, and constitutes one of the most important signs of brain tumor. Amblyopia, defective vision or blindness may accompany the choked disk. The field of vision may be contracted with inversion of the color-fields, so that the field for blue may interlace with or lie within that for red.

FOCAL SYMPTOMS.—These depend upon the location.

Frontal Region.—Changes in temperament and mental activity are notable. Memory is impaired, ability to concentrate thought or to learn diminishes, and the patient becomes stupid and irritable, or uncontrollable.

Tumors of the third left frontal convolution in right-handed persons cause motor aphasia with agraphia. In left-handed persons the tumor must be in the right hemisphere.

Motor Area.—Tonic or clonic spasms of the muscles of some portion of the body are caused by tumors of the motor cortex. The spasms so caused occur at intervals and may terminate in general convulsions. The spasms always begin in those muscles whose centers of

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representation are most affected by the tumor. Therefore, observation of these spasms as to their location at the beginning and the mode of spreading helps greatly in locating a tumor. Either extremity or the face may be affected. Paralysis or paresis may follow either in arm, leg or face, but cortical tumors rarely produce hemiplegia. Tumors involving the internal capsule or the motor tract beyond it may cause hemiplegia.

Parietal Region.—Disturbances are not constant, but in many cases impairment of sensation or muscular sense follows. Word-blindness has been found to be due to lesion of the inferior parietal lobule (left).

Occipital Region.—Hemianopsia (bilateral homonymous) results from lesion of the occipital lobe. It may be unknown to the patient and must be tested for.

Island of Reil.—Tumors of this area produce paraphasia—that is, disturbance of speech in which one word is regularly substituted for another. They may produce pressure upon the neighboring face centers or even upon the internal capsule.

Crus.—Tumor involving the crus should produce paralysis of the third nerve of the same side and the extremities of the opposite side of the body. The fourth nerve may also be involved.

Pons.—Tumors in the upper part involve the third and fifth, in the lower part, the fifth, sixth, seventh and eighth. In this situation paralysis of one or more of these nerves with loss of power on the opposite side of the body, so-called alternating hemiplegia, may result.

Medulla.—The ninth, tenth, eleventh and twelfth nerves suffer with resulting difficulties in swallowing, in respiration, irregularity of the pulse, and paralysis of the tongue. Polyuria or glycosuria may also result.

Cerebellum.—Because of the small space in which the cerebellum lies enclosed by the tentorium cerebelli, small tumors produce severe results. By compressing the fourth ventricle, tumors in this region cause distention of the ventricles above, and hence symptoms appear early. Staggering gait, vertigo and cerebellar ataxia, an incoördination which entirely disappears when the patient lies down, are marked. Nystagmus is frequently present. Knee-jerks are exaggerated.

Diagnosis.—The symptoms of steadily increasing intracranial pressure with the presence of choked disk are characteristic. Abscess of the brain must be excluded by the absence of any cause for suppuration, absence of any constant leukocytosis, or fever, or other constitutional symptoms of suppuration and the presence of optic neuritis. Brain abscess rarely gives rise to definite focal symptoms and then only late in the disease.

Tubercular meningitis causes a more rapid development of symptoms, with hyperesthesia to light or sound, possibly tubercles in the chorioid, and without optic neuritis. Tubercle bacilli should be found in the spinal fluid. Chronic hydrocephalus in children is excluded by the size and shape of the head, the symmetry of the rigidity or loss of power, absence of choked disk, and the comparative comfort of the patients.

The Wassermann reaction is a valuable aid in the diagnosis of gumma.

Course and Prognosis.—Gradual but steady increase in symptoms over one or two years is the usual history. Death at the end of two or three years results in nearly all cases. Gummata may be relieved, possibly cured by specific treatment. A few tumors have been successfully removed by surgery, but only ten per cent. are operable, and of those operated upon but few survive.

Treatment.—Active antisyphilitic treatment should be tried. The iodide of potassium is given in increasing doses up to 60 grains thrice daily. Inunctions of mercurial ointment or injections of mercury may be combined with the iodide. Careful study should be made in the effort to locate the tumor and operation undertaken, if it is accessible.

APHASIA

Definition.—Literally the word means loss of the power of speech, but in medicine includes all the disturbances of the use of language, either written or spoken, not dependent on intellectual failure or paralysis of the vocal organs. Aphasia is a symptom of various cerebral lesions, not a disease in itself. The exact determination of the defect in speech is of material aid in the location of cerebral lesions, especially brain tumors. The subject is, therefore, of sufficient importance to be dealt with separately.

To understand the different types of aphasia one must go back to the mental processes underlying the use of words in speaking or writing. Every word has four points of relation or centers in the cerebral cortex. It can be heard or seen, it may be spoken or written. Thus the auditory center lies in the temporal lobe, the visual center in the angular gyrus, the motor speech center in the third left frontal convolution, and the center for writing in close relation to it in the centers for arm and hand. Each of these actions depends upon the integrity of a definite area of the cortex, and also the normal use of words in speaking and writing depends not only upon integrity of these centers, but on their free communication with one another through the association fibers of the brain.

The chief types of aphasia are described thus:

1. SENSORY APHASIA.—1. *Word-Deafness.*—This condition is best illustrated by the common experience of forgetting names. Every one has at some time seen an acquaintance whom he sees, recognizes, perhaps knows all about, and yet cannot name. The memory picture of his name cannot be recalled and hence cannot be expressed. But if now the acquaintance says my name is John King, the recollection may still fail, if the defect is complete, but usually the sound of the name at once recalls its word picture and one recognizes its propriety and utters it. The condition here described is, of course, a matter of momentary forgetfulness, but in true word-deafness the condition persists. The patient is therefore unable to name familiar objects when

brought to mind in any manner. In this condition there must be a cortical lesion involving the sound memory centers in the temporosphenoidal lobe. If the defect is but partial, the name of a thing cannot be recalled of one's self, but is recognized at once when heard. This auditory amnesia, as it is sometimes called, implies the integrity of the word-centers, but a defect, sub-cortical, in the fibers connecting them with other centers. This distinction between cortical and sub-cortical lesions is of importance mainly with reference to the possibility of operating upon cerebral lesions such as tumors.

2. *Word-Blindness.*—If this condition is complete word pictures are entirely lost. The thing seen is not recognized, and hence cannot be named. If the defect is partial only, the missing word picture, not reached by one route, the eye for example, can at once be recalled if the approach is made through another channel such as the ear. Thus a patient with complete word-blindness cannot recognize and name a bell, for example, when seen, but if it is rung in his ear he recognizes it at once and on request draws it in outline. He has the concept of a bell with its form, but it cannot be reached through the visual route.

With relation to language complete word-blindness is shown by inability to read or write. The patient cannot read because he cannot recognize the characters before him. He cannot write because he cannot recall the shapes of the necessary letters. If the defect be only partial, he can write at dictation or copy, although not understanding what is written.

Complete word-blindness indicates a cortical lesion in the region of the inferior parietal convolutions and angular gyrus. A partial word-blindness is produced by a sub-cortical lesion involving the fibers of association.

3. *Intercortical Sensory Aphasia.*—The fibers of association between the auditory and visual word centers may be interrupted, producing a condition in which an object seen cannot be named, or if named cannot be recalled to mind, although recognized in both instances. This condition is produced by lesions of the association fibers connecting the temporal and occipital lobes.

2. MOTOR APHASIA.—1. *Aphemia,* or what is commonly called simply aphasia, a condition in which although words or things are recognized by eye and ear, the patient cannot name them or speak because of inability to recall the muscular movement necessary. This happens, of course, independent of paralysis of the muscles of speech. 2. *Agraphia,* the condition in which one can name objects and speak as usual, but cannot write, because the memory of the movements necessary is lost. 3. *Intercortical motor aphasia or paraphasia.* In this condition an interruption or interference with the association fibers connecting the motor and other word-memory centers results in a disturbance characterized by the use of the wrong word in speech or writing. The patient speaks or writes jargon. Paraphasia is particularly associated with lesions of the island of Reil.

AMAUROTIC FAMILY IDIOCY

Definition.—A rare affection of Hebrew children, characterized by mental impairment during the period of infancy, blindness and loss of power over much of the body.

Etiology.—The causation is unknown. The disease occurs only among the Hebrews, and often affects more than one child in a family. It is not syphilitic.

Morbid Anatomy.—A degenerative process is found affecting all the nerve-cells of the brain and cord. The degeneration affects particularly the body of the cell and the dendrites, not the axis cylinder. The changes in the cell may be slight or so complete that the cell is disintegrated. Sachs thinks the disease due to congenital deficiency of the nervous system, such that after three or four months of normal life, the cells yield to the ordinary demands of life and disintegrate.

Symptoms.—These are briefly summed up by Sachs as: (1) Mental impairment during the first few months of life leading to complete idiocy. (2) Paresis or paralysis, either flaccid or spastic, of the greater part of the body. (3) The reflexes may be deficient, normal or increased. (4) A diminution of vision terminating in absolute blindness. (5) Marasmus and death before the age of two years.

Various other symptoms, such as increased sensibility to touch or sound, nystagmus, strabismus, or convulsions, have been noted in some cases.

Diagnosis.—This rests upon the mental impairment, the blindness and the ophthalmoscopic examination, which shows, in the region of the macula lutea, a bright cherry-red spot, an absolutely pathognomonic sign. (See Plate VII.) Treatment is of no avail.

FUNCTIONAL NERVOUS DISEASES

ACUTE DELIRIUM

(Typhomania. Brain Fever. Bell's Mania)

Definition.—An acute delirium or mania accompanied by fever, regularly fatal, without adequate organic lesions.

Etiology.—Emotional excitement, injury, toxemia and infection are given as causes. In most instances no satisfactory explanation is to be had.

Morbid Anatomy.—The meninges are congested, but the brain appears normal. Microscopically Osler found some exudation of leukocytes about the vessels and in the lymph-spaces.

Symptoms.—The onset may be preceded by several days of restlessness, irritability and insomnia. The onset is usually marked by fever, 102° to 104° F., with some increase in the pulse and prostration. Wild delirium or an active mania develops easily. Hallucinations are vivid, the patient talks or sings incessantly, and keeps in constant motion.

Such acts as salaaming may be repeated endlessly. The patient may struggle with his attendants, but rarely injures anyone but himself.

A typhoid-like condition is developed with fever, a dry-brown tongue, rapid pulse, marked prostration, and rapid emaciation. At the end of a week or ten days the patient dies in collapse.

Diagnosis.—Typhoid fever must be excluded by absence of the characteristic roseola and the enlargement of the spleen, and by the study of the leukocyte count, the Widal reaction, and blood cultures. Pneumonia can be excluded only by the absence of the usual symptoms and repeated careful physical examinations. Delirium tremens presents a distinct etiology, characteristic hallucinations, and the active tremor of tongue and hands.

Acute meningitis may be excluded by the absence of rigidity of the neck and Kernig's sign, and by the results of lumbar puncture. It is evident that the diagnosis of acute delirium can hardly be safely ventured before the autopsy reveals the absence of lesions sufficient to account for the disease.

Treatment.—Blood-letting has been recommended in the early stages. The general treatment must be that of typhoid fever. The bromides gr. xxx t. i. d. may be given for excitement or hyoscin in doses of 1/100 gr. Dram doses of the fluid extract of ergot every two hours are recommended.

PARALYSIS AGITANS

Definition.—A chronic nervous affection marked by muscular weakness, tremors and rigidity.

Etiology.—Two-thirds of the patients are men. The disease rarely begins before forty. Exposure, privation and nervous strain appear to favor its development. Numbers of cases were observed during the siege of Paris and the war of the rebellion.

Morbid Anatomy.—No distinctive lesions have been found. Brawny patches in the skin have suggested myxedema, and the theory that lesions of the parathyroids were related to the disease has been advanced.

Symptoms.—The onset is either sudden or gradual. Usually the weakness, stiffness and tremor appear in one hand or arm, then the other. All four extremities and even the head may be affected. When fully developed the clinical picture includes:

1. The tremor, the most striking symptom. It is a rapid tremor, four to eight oscillations per second, usually most marked in the hand. The movement of the fingers suggests pill-rolling. Flexion and extension of the wrist or slight rotation may be combined with the finger movements. It may show clearly in the hand-writing. It usually is present at rest, and disappears on effort, but in some cases accompanies voluntary action—*i.e.*, is *intentional*. In the foot the tremor causes rapid flexion and extension at the ankle, and the toe taps the floor as in clonus.

2. Rigidity of the muscles is the most pronounced feature of the disease. Rigidity precedes or accompanies the tremor. Passive motion may be impeded, but the reflexes are not increased and ankle clonus is not found. The facies becomes mask-like. The eyes are held open and fixed in one position, the whole head being revolved instead of moving the eyes from side to side. The patient rarely winks. The neck muscles are rigid as in torticollis, but both sides are involved, and the face is not turned to either side. The body is bent forward at the hips and the spine is bowed forward. In the hands similarly the fingers and wrists are usually flexed. Speech and gait are often characteristic. In speaking the patient hesitates a moment, then hurries the words out and stops abruptly. In like manner the patient, rising from a chair, halts a moment, then with the body bent forward advances with short, hurried steps, as though the feet were trying to overtake the body. In rare cases the patient actually falls forward.

3. The mental state is marked by inertia. The patients are dull, sluggish, talk little and are indifferent. Dementia is often suggested, but when roused the patients show complete possession of their faculties.

4. Sensory disturbances. The patients complain of sensations of heat or cold, but respond normally to tests. Vasomotor changes may be indicated by flushing of the skin, increased sweating or salivation.

5. Muscular atrophy is not marked and no electrical changes are present.

Course.—The disease is chronic and incurable, lasting many years. Periods of improvement may occur.

Diagnosis.—In a typical case the diagnosis can be made on sight. The attitude of the body, the gait, the mask-like face, and muscular rigidity as well as the tremor are characteristic. Disseminated sclerosis is marked by earlier onset, scanning speech, and nystagmus. The tremor is regularly intentional. Post-hemiplegic tremor can be excluded by the history of preceding paralysis and the increased reflexes. Hysteria must be considered.

Treatment.—Medication appears to be of no avail. Massage, electricity and vibration have been of service. The administration of extracts of the parathyroid glands has been advised.

ACUTE CHOREA

(Chorea Minor. Sydenham's Chorea. St. Vitus' Dance)

Definition.—A nervous affection, common in children, characterized by irregular involuntary muscular contractions, resulting in purposeless movements, and often accompanied by psychic disturbances.

Etiology.—The disease is common between the ages of five and fifteen, especially in girls. A neurotic heredity can often be traced. The strain of school-life is usually the chief factor. Sudden fright or ex-

citement often precedes the appearance of symptoms. About 25% of the patients give a history of antecedent rheumatism. Any acute infectious disease or other condition lowering the vitality of a child may be an accessory cause of chorea.

Morbid Anatomy.—Death from chorea is rare and pathological studies relatively few. Minute emboli in the cerebral vessels have been found in some cases, and have led to the theory that chorea is caused by numbers of such emboli taking their origin from a rheumatic endocarditis. This theory covers but a part of the cases.

Perivascular hemorrhages and areas of infiltration in the brain have also been observed. The pathology is by no means established.

Bacteria.—Many investigators have found bacteria in the brain, but the organisms have been of wide variety, usually streptococci. Poynton and Paine found their streptococcus rheumaticus in the cerebral embolisms. The dependence of the disease upon bacterial infection is not established, but the theory that it is due to the action of toxins, probably bacterial, upon a sensitive nervous system receives much support.

Symptoms.—Motor.—Irregular, jerky, purposeless movements of one or both upper extremities are first noted. The movements may be confined to one extremity or extend to all. The face may be affected. With these movements awkwardness and weakness in the use of the hands or other parts develop. The patient drops things because of the chorea and is often rebuked or punished for carelessness. Speech may become hesitating, jerky and imperfect. The movements may be very slight, occurring at rare intervals and for some time scarcely attracting attention. In the mild form they cease during sleep. They are regularly increased by excitement or embarrassment, and are therefore often most noticeable during examination. In severe cases the jerky movements affect all parts of the body, are almost constant, more violent, and may persist during sleep. The weakness may develop into paralysis.

Psychic.—Irritability, peevishness and inability to concentrate the attention attend the milder cases. In severer forms the mental disturbance becomes more marked. Delirium with varied hallucinations may develop. Stupor and dementia have been observed in fatal cases. Constitutional anemia, malnutrition and loss of appetite are regularly present. The pulse is rapid and the patients feeble. Fever appears only in the severer cases.

Complications.—Heart murmurs are common. They may be hemic or due to definite endocarditis. The location of the apex impulse, the size of the heart, the character of its action, and the circulatory conditions must all be considered in attempting to decide this question. Acute pericarditis occasionally develops. Other rheumatic complications, such as erythema nodosum, purpura and subcutaneous nodules, may be seen.

Course.—The disease runs its course in two or three weeks or

months. It regularly ends in recovery. Occasional choreic movements may be seen for months afterward. Death is possible in the severe forms.

Treatment.—Freedom from disturbing influences of any kind, fresh air and good food are the prime requirements. The home conditions must be made favorable or the child removed from them. Relief from school duties, an out-door life, and nutritious food will promptly relieve mild cases. In pronounced cases rest in bed is necessary.

Arsenic is regularly administered. Fowler's solution in doses of 2 to 5 minims, thrice daily, is commonly given. Some increase the dose to the limit of intolerance, but this seems undesirable.

Chloral and the bromides are often given for sedative effect. Massage and passive movements may be employed. In chronic cases suggestion and educative exercises are of value.

CONVULSIONS OF CHILDREN
(Infantile Convulsions)

Definition.—General tonic and clonic convulsions occurring in childhood from various causes, not including epilepsy.

Etiology.—A family tendency to nervous disturbance is notable in some cases; in others the individual seems to have an abnormally sensitive nervous system. The origin of this weakness can often not be made out. In children of such hereditary or acquired nervous susceptibility any form of trauma, toxemia or infection may be the cause of a general convulsion. The possible causes of convulsions in childhood, therefore, include practically all the varied forms of disease or injury to which they are subject. Certain influences are so important as to be specially enumerated. 1. Gastro-intestinal disorders of any kind, especially overloading the stomach with indigestible food, and constipation, are the commonest causes of convulsions in childhood. Intestinal parasites and inflammations of any kind are important factors.

Dentition is commonly regarded as the most frequent cause of infantile convulsions. In most instances it is some associated alimentary disorder and not the "cutting of the teeth" that causes the disturbance, yet in very sensitive children "teething" alone may cause convulsions.

2. In childhood the invasion of an acute infectious disease, especially pneumonia, scarlet fever, whooping-cough or small-pox, is frequently marked by a convulsion. The initial chill commonly seen in adults at the onset of such acute infections seems to be replaced in childhood by a convulsion.

3. Rachitis predisposes children to convulsive seizures.

4. Any illness or indisposition lowering the vitality of a child favors these convulsive seizures.

5. Fright or excitement may precipitate a convulsion in a susceptible child.

Symptoms.—The convulsions may occur in a child apparently in perfect health or they may be preceded by irritability and restlessness with evidences of digestive disturbance.

The seizures vary from attacks of brief unconsciousness with little or no spasm to general tonic and clonic convulsions not to be distinguished from the characteristic fits of epilepsy.

In the milder attacks, the so-called ''inward spasm,'' the child becomes silent, rigid, stares or rolls the eyes upward, the face or one hand twitches for a moment and the paroxysm is over.

The severer paroxysms begin in like manner, but the whole body becomes rigid, the hands clinched, the elbows flexed, the head retracted, and quick spasmodic jerkings of the face and extremities follow. The respiration is embarrassed, the pulse becomes rapid, feeble, and possibly imperceptible. The face and lips become deeply cyanosed and death appears imminent. There may be frothing at the mouth and rattling of mucus in the throat. After lasting a few seconds or several minutes the convulsion gradually ceases, the muscles relax, and the child passes into a stupor from which it rouses slowly, perhaps hours afterward. The attacks may be repeated at any time; frequently several follow one another at short intervals, and many may occur in a day. Death rarely follows a single convulsion, but may result if the convulsions are frequently repeated.

The convulsions are likely to recur, but may not. In most cases they cease entirely as the child grows older. Persistence of the convulsions usually means epilepsy.

Diagnosis.—The convulsion can be recognized on sight or history. The cause is the important question and must be sought with care. A thorough physical examination and a study of the diet and life of the child should be made to determine the conditions which have caused the convulsions. Epilepsy can be excluded only after prolonged observations, but it is to be remembered that convulsions are common, epilepsy in infancy relatively rare.

Treatment.—During the convulsion chloroform should be given by inhalation. If the tongue is protruded or caught between the teeth a bit of wood or rubber should be used as a gag to prevent injury to it. A hot bath or hot pack, with mustard added to the water in the proportion of a tablespoonful to the gallon, should be given at once with the object of reducing internal congestion by bringing the blood to the surface. The bowel should be emptied by an enema, and castor oil given by mouth. Chloral and the bromide of soda may then be given by rectum, 5 grains of chloral and 10 of the bromide to a child of a year or more. If the danger of recurrence seems great, morphine sulphate, gr. 1/50 for a child of one year, may be given hypodermatically.

Quiet and a restricted diet should be required for several days before allowing return to the ordinary mode of life. Careful examination should then be made for any possible cause of convulsions. The diet and regulation of the life are of prime importance. Rachitis if

present must be treated. Adenoids may require removal. Anemia should be treated, and in every way the general health of the child maintained at the highest point. Excitement or excess of any kind must be avoided. If under such care convulsions are repeated the probability of epilepsy is increased.

EPILEPSY

Definition.—A nervous affection characterized by periodic attacks of unconsciousness commonly associated with general convulsions and frequently preceded by an aura.

Etiology.—Epilepsy regularly develops in childhood in either sex, and rarely begins after the age of thirty. Heredity plays an important part. If epilepsy itself is not present in the family, neuroses of some kind are often found.

Syphilis, tuberculosis or alcoholism in the parents predisposes to epilepsy. Every influence which lowers the nervous vigor of a family favors the development of epilepsy. Similarly any influence which lowers the nervous vigor of the individual may in a susceptible person precipitate the first attack or cause the repetition of the convulsions. (a) Toxic influences, such as alcohol, tobacco, or the toxemia resulting from overburdening the alimentary tract and constipation, are of first importance. (b) Acute disease of any kind, especially the acute infectious diseases, may precede the onset. (c) Reflex irritations from eye-strain, adenoids, intestinal parasites, uterine or ovarian disease, phimosis and the like. (d) Mental shock or excitement are important agencies. (e) In females the attacks are likely to occur at the time of menstruation.

The cerebral hemorrhages of birth, injuries to the skull at that time or later, defective cerebral development from any cause, all favor epilepsy. Idiocy or imbecility and epilepsy are frequently associated.

Morbid Anatomy.—Many brain lesions have been found in patients suffering from epilepsy, especially defects of development, porencephalus, hemorrhage, cysts, tumors, meningitis and the like. These lesions are not essential, however, but may be regarded as sources of irritation which favor the development of convulsions. Recently careful studies have shown that in those dying of epilepsy degenerations in the cerebral nerve-cells, especially those of the second cortical layer, can be demonstrated. These degenerations involve especially the nucleus and nucleolus.

Epilepsy is still, however, regarded as a functional disease—*i.e.,* without definite anatomical basis.

Symptoms.—1. GRAND MAL.—The seizures of general convulsions are the typical form of the malady. These include a tonic stage, a clonic stage, and a period of stupor or coma, and in many cases are preceded by an aura.

The Aura.—A warning of some kind occurs in many cases; it may be motor, sensory or psychic. Motor. A tremor, a jerk or movement of

some part or parts of the body, rapid revolution, or running forward a few steps are sometimes seen. Sensory auræ are more common. Tingling, burning, numbness, a certain sound, a flash of light and various other unusual sensations are recorded. Psychic auræ present themselves as a certain mental depression or exaltation, certain emotions, or recollections. Whatever the aura it is repeated, as a rule, before each fit. The aura may just precede the paroxysm, but sometimes an interval of some minutes elapses before the onset. Auræ are present in a majority of patients but not in all.

Tonic Stage.—The patient suddenly becomes unconscious and falls, often injuring himself about the head and face in falling. The whole body becomes rigid. The head is drawn back or to one side, the eyes are turned upward and to one side, the face is pale, the thorax immovable (apnea), the limbs flexed. Often the sudden contraction of the respiratory muscles causes an inarticulate cry. The tonic stage lasts but a minute or two.

Clonic Stage.—The convulsive movements begin in the face or an extremity, but quickly become general. The eyes, face, limbs and whole body are thrown into violent jerky contractions, the teeth are ground together, the tongue is protruded and often bitten, the saliva is churned into froth and pours from the mouth, the respiration is jerky and imperfect, the face becomes cyanotic, the pulse rapid and weak. The contractions last from one to five minutes, and then gradually die out. Both bladder and rectum are commonly evacuated during the paroxysm. Stupor or complete coma follows, lasting a half hour or more. The patient then awakes, possibly unconscious that he has had a convulsion, but usually weak, depressed and mentally confused.

Recurrence.—Epileptic fits recur after varying intervals, hours, days, months or years. The fits often occur in groups. Fright, fatigue, excitement and the like may precipitate an attack, but usually they recur without apparent cause. In most cases the attacks slowly increase in frequency.

Nocturnal epilepsy is common, the fits coming on at any time during the night, even in sleep. Under these circumstances the patient may not know what has happened or may be aware from the muscular soreness, exhaustion or the bitten tongue that he has had a fit.

Status Epilepticus.—In some instances, especially late in the disease, the fits are repeated in a long series, are accompanied by fever, even 104° to 105° F., and followed by collapse. Such crises end favorably, as a rule, but death may occur.

Postepileptic State.—Usually the mind quickly clears, but it may remain clouded. A trance-like condition may supervene, and mania with homicidal tendencies is well known to follow in some cases.

2. Petit Mal.—Temporary unconsciousness with few or no convulsive signs marks this condition. The manifestations are varied. In some a sudden pallor, a fixed stare, a momentary obliviousness, are all that are seen. Slight tremor of the eyes, face or an extremity may be seen.

The patient may fall or may drop what he is holding. The urine may be passed involuntarily.

In other cases the patient turns rapidly about, or runs a few steps in an automatic manner, and then returns to his usual condition.

Psychic Equivalents.—In rare cases the epileptic seizure is replaced by some unconscious and automatic act. Murder has been done, and fires set by the irresponsible victims of such seizures. In other cases long journeys have been taken or various complicated acts performed. Ordinary convulsions occurring before or after such paroxysms reveal their nature.

Course.—The disease tends to grow worse with steady increase in frequency of the convulsions and increasing mental dulness in the intervals. The patients may die from their falls or injuries received while unconscious (drowning) or from the convulsions, or in a condition of dementia.

Diagnosis.—The typical fit can hardly be mistaken. The aura, tonic and clonic spasm and subsequent stupor are very characteristic. Repetition of the convulsions is highly suggestive. The presence of scars or bruises on the scalp or the tongue is helpful. Nocturnal fits are regularly epileptic. In children under two years convulsions are often repeated a number of times and then cease, without subsequent epilepsy. The family history is important, but time is required to settle the question. In adults the convulsions of uremia are sometimes mistaken for epilepsy. The anemia, cardiac changes, arteriosclerosis and urinary findings should differentiate the condition.

Hysteria is easily recognized. The fit is overdone. The patient is noisy, never injures himself or herself, but may strike others; the convulsion is exaggerated, unconsciousness is feigned, but there is no relaxation of the sphincters and no coma.

3. JACKSONIAN EPILEPSY.—(Cortical, symptomatic or partial epilepsy.) In this condition the seizures are limited to one extremity or to one side of the face. The convulsion may extend from one part to another, as from the arm to the leg or vice versa. The localized form may at any time give place to a general convulsion. Jacksonian epilepsy usually arises from focal irritation of the brain, such as a depressed fracture, a localized meningitis, a tumor or abscess. Similar attacks may occur in uremia. The location of the focal lesion is usually indicated by the part in which the convulsion begins.

Prognosis.—Some few patients recover, but the disease is generally incurable and tends to be progressive.

Treatment.—GENERAL.—The patient must be examined thoroughly for any defect which may be a source of irritation. Eye-strain, adenoids, uterine displacement, any possible source of local irritation should be relieved. The digestion should be studied, an easily digestible diet prescribed, and constipation avoided. An out-door life with freedom from annoyance or care is helpful. Moderate exercise is desirable. Excess of any kind must be forbidden.

MEDICINAL.—Bromide of potassium or sodium is effective in reducing the frequency of the paroxysms. Small doses, 5 grains, are given, and the dose later increased. If acne appears, the medicine must be reduced or stopped. It may be continued indefinitely. Many other nerve sedatives, chloral, cannabis indica, zinc, etc., are employed.

INSTITUTIONAL.—Control of the mode of life of the epileptic patient is essential. If this cannot be secured at home, removal to a sanatorium should be urged. Great numbers of epileptic patients are now treated advantageously in farm-colonies, such as the Craig Colony, Sonyea, N. Y. Jacksonian epilepsy may be relieved by trephining and appropriate treatment of the focal lesion.

MIGRAINE
(Megrim. Sick Headache)

Definition.—An affection characterized by attacks of severe headache, often attended by disturbances of vision and digestion. Migraine presents a close analogy to an epilepsy in which the motor discharges are replaced in large part by sensory phenomena.

Etiology.—The explanation of migraine is regularly based on two factors, a constitutional nervous defect or weakness, and some local disturbance or weakness. (a) Constitutional. Heredity is regarded as important. Some neuropathic taint (headaches, epilepsy, neurasthenia and the like) can usually be found in the family. A gouty tendency is sometimes present and excess of uric acid in the blood or deficiency of it in the secretions is commonly invoked as explaining the attacks of megrim. Exact proof of the relation of either of these conditions to megrim is, however, lacking, and the common accusation of uric acid in this regard is without adequate foundation.

(b) Local conditions. These comprise most functional and organic defects, such as simple constipation, eye-strain, adenoids, nasal spurs and polypi, uterine or ovarian disease and the like. In general any influence which lowers the health of the patient or acts as a source of local irritation may have relation to attacks of migraine. In many cases it is quite impossible to determine the etiology. The affection appears in families without neuropathic taint and in healthy individuals. The attacks begin in childhood or early adult life, more often in females.

Symptoms.—Prodromal disturbances announce to some the approaching attack. The prodromata are varied, languor, drowsiness, flashes of light, spasm of ocular muscles, dilatation of the pupil and the like. The headache itself usually begins in the morning. The pain is referred to any part of the head, usually unilateral, sometimes sharply localized. Visual disturbances commonly accompany the pain. The sight is blurred, or there may be definite hemianopsia.

In some cases varied figures with flashing colors play before the eyes. Nausea and vomiting are common; the vomitus is yellow from the presence of bile. Vomiting is severe in some persons and may give relief.

Tingling and numbness may be felt in the face or arm, and may be accompanied by weakness or actual loss of power. Speech may be slow and stumbling and temporary aphasia may develop. The face is usually pale from vasomotor constriction, and later the face and ear on the affected side may flush. The patient is greatly prostrated and hypersensitive to noise or disturbance of any kind. The attack usually lasts throughout the day or several days. The patient is prostrated for some time afterward.

Course.—The attacks occur at intervals of days or weeks, sometimes from definite indiscretions in diet or life, but often without apparent reason and despite the best of treatment. They tend to become less frequent and less severe in later life. The menopause seems to bring relief to women. Many patients having sought relief in vain finally resign themselves to their suffering, and yet are able to live lives of activity and usefulness.

Treatment.—THE ATTACK.—A brisk saline cathartic taken at the first signal of the attack is advisable. Washing out the stomach with hot water is recommended in those cases where vomiting is severe. Black coffee given at the onset is sometimes helpful. During the attack the patient must be kept in bed and heat applied to the feet. Acetanilid, phenacetin and caffeine are given for relief of the pain, and are sometimes helpful, if they can be retained. Morphine may be required for the very severe attacks, but should be withheld, if possible. Often the after-effects of morphine are so disagreeable to these patients that its relief is declined. Hot or cold applications, as preferred, may be made to the head.

GENERAL.—In the intervals the life of the patient should be carefully planned. A vegetarian diet helps some, but dietary restrictions are usually valueless. Avoidance of such articles of food as cause gastric disturbance usually commends itself to the patient. Excesses of any kind should be avoided. Abundance of sleep and rest is advisable. An out-door life and moderate exercise are recommended.

LOCAL.—Every defect should, if possible, be corrected. Thus errors of refraction, adenoids, bad teeth, digestive disturbances, especially constipation, uterine or ovarian disease and the like must be appropriately treated.

NEURALGIA

Definition.—A painful affection of a nerve or nerves without discoverable lesions either in the nerve itself or adjacent tissues to explain it.

The term is a broad one which commonly is loosely used to cover all types of pain. Every effort should be made to distinguish neuralgia, on the one hand, from pain excited by definite nerve changes such as occur in neuritis, and on the other from pain due to definite local lesions, such as abscesses, new growths and the like. Even with the most careful study, however, we may be unable in some cases to distinguish neuralgia from neuritis.

Etiology.—The affection is rarely met with in childhood, but is common among adults, especially women. A neurotic tendency or heredity underlies many cases. The weak and anemic are most subject to it. Constitutional conditions producing weakness and anemia may precede it, especially infectious diseases, such as influenza and malaria, or the systemic poisons of rheumatism, gout, diabetes or chronic nephritis, or alcohol, lead or arsenic. In those liable to the affection, exposure to cold and wet, or any condition producing nervous exhaustion or debility may precipitate an attack. In not a few cases no cause can be found and we must be satisfied to call them idiopathic. The term ought not to be applied to pains which can be explained by definite pathological lesions, such as caries of the teeth, suppuration in the antrum of Highmore, pressure of tumors and the like. The more carefully patients are studied the less use we shall have for this designation.

Symptoms.—Neuralgia presents itself in the form of paroxysms of pain, usually described as sharp, burning or stabbing, in the territory of a certain nerve or nerves of one side of the head or body. The paroxysms last for a brief time or may be protracted. In the intervals there is usually no discomfort. The pain is usually severe, and may be agonizing. Regularly there are no changes seen in the affected area. There may, however, be vasomotor or finally trophic changes in persistent cases. Thus the skin may be hot and red, or cold and pale. Edema, atrophy or induration of the skin, or whitening of the hair in chronic cases may be seen. Where herpes or such changes as these are met with we are probably dealing with manifestations of definite lesions of the nervous system. (See Herpes Zoster.)

The attacks of pain are likely to recur at regular intervals of a day or days, without relation to malaria. The pain often shifts from one nerve to another. Along the course of the affected nerve there may be tender points, especially where it emerges from a canal or becomes more superficial. The affection lasts for varying periods, weeks or months, or in some cases persists for life. Any nerve in the body may be affected. The more important varieties follow:

(1) TRIGEMINAL NEURALGIA. Tic Douloureux. Any of the branches of the fifth nerve may be involved. The attacks of pain are characteristic. The affection is often severe and protracted, and may require heroic measures for relief. Before accepting the diagnosis of neuralgia we must carefully exclude organic lesions, such as the pressure of tumors, cerebral or in the course of the nerve, disease of bone causing pressure in the course of the nerve, or any cause of peripheral irritation, such as caries of the teeth, suppuration of the antrum or the frontal or ethmoidal sinuses, or the growth of tumors within the orbit, nose or mouth. In neuralgias of the ophthalmic division the possibility of glaucoma, or errors of refraction or muscular defects of the eyes must be considered.

(2) INTERCOSTAL NEURALGIA.—This is frequently a severe type of the affection. Care must be taken to exclude pressure on the nerve by diseased vertebræ (Pott's disease) or aneurisms or other tumors. Pleurisy

ing of the normal side for pathological. Much less frequently the breath sounds may be harsh or broncho-vesicular. Often the respiratory murmur is interrupted or wavy (cog-wheel breathing). The voice sounds usually show no appreciable change. A few fine râles are frequently heard over the affected area and are of much significance. They may be due to the localized bronchitis, to the pleurisy or to beginning softening. A short, sharp cough followed by a quiet inspiration will often disclose the presence of râles not heard under ordinary conditions. For their detection and localization the stethoscope is to be preferred to the unaided ear.

SIGNS OF THE ADVANCED STAGE.—INSPECTION shows emaciation of varying degree, anemia, increase in breathing rate, pronounced retraction above and below the clavicles, restricted chest expansion and often a much increased area of cardiac pulsation. PALPATION verifies the restriction of chest movement and reveals increased vocal fremitus over areas of consolidation or diminished fremitus over thickened pleura or pleuritic effusion. Friction fremitus, or that corresponding to coarse râles or rhonchi, may also be felt. PERCUSSION gives dulness of varying degrees, corresponding to the extent and completeness of the consolidation. It also often shows immobility of the lung borders, especially over the liver and heart, which indicates pleuritic adhesions. A large and superficial cavity may give a tympanitic or amphoric note or the cracked-pot sound. Usually, however, the cavities are so surrounded by consolidation, or are so filled with secretion, that they give no characteristic percussion note. Over areas of complete consolidation AUSCULTATION shows tubular (bronchial) breathing and bronchophony. In other places the breathing may be broncho-vesicular; in still others it may be greatly diminished, because of fibrosis or from pleuritic adhesions. Râles are usually abundant and may be of all possible varieties, from the fine crepitant râles of hepatization to the loud, coarse, gurgling sounds of a cavity containing secretion.

A CAVITY does not often show characteristic signs. This may be due to its small size; to its being deep in the lung, or surrounded by consolidation, or filled with secretion, or to the fact that its communication with a bronchus is not free. Usually, however, the breathing over it possesses the exaggerated, hollow, tubular quality known as cavernous. If this has beside a musical quality it is called amphoric. The voice also has the same cavernous character and the whisper is heard with astonishing distinctness and loudness—as though through a trumpet (whispering pectoriloquy). The characteristic râles of a cavity are very coarse, loud, gurgling sounds which have a peculiar resonant quality. PALPATION may reveal absence, diminution or increase of the vocal fremitus, or the crepitation of coarse râles.

Complications.—PLEURISY with effusion is common. The fluid may be serous, hemorrhagic or, rarely, purulent.

PNEUMOTHORAX occurs occasionally—usually by the perforation of a small, superficial cavity. Tuberculosis of the larynx, and of the intes-

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tines, develops at some time in about half of all cases. The disease may attack the meninges, brain, peritoneum, pericardium, kidneys, bladder, liver, etc. COMPENSATORY EMPHYSEMA is very common in the unaffected portions of the lungs. LOBAR PNEUMONIA or PULMONARY GANGRENE may occur. Other complications are FISTULA IN ANO, dilation of the stomach, venous thrombosis and endocarditis. Although actual endocarditis is comparatively rare, heart murmurs are by no means uncommon. They are systolic in time and are located either at the apex or over the pulmonic area. Retraction of the lungs from fibrosis often causes great increase in area of cardiac pulsation and sometimes actual displacement of the heart.

Diagnosis.—The importance of a diagnosis at the earliest possible moment cannot be overestimated. The history of the case, the symptoms, and the physical signs should be carefully studied and weighed. The sputum should be examined, repeatedly, if necessary, for tubercle bacilli, the presence of which furnishes the only absolute proof of the disease. Failure to find the bacilli does not prove conclusively the absence of tuberculosis. The presence of elastic fibers in the sputum is evidence of the destruction of lung tissue, but these are found in other conditions than tuberculosis—*e.g.*, pulmonary gangrene.

OTHER DIAGNOSTIC AIDS.—*I. Tuberculin.*—A variety of preparations derived from tubercle bacilli are now used under the name of tuberculin. The best known and most commonly employed of these is that first prepared by Koch and now known as old tuberculin or T.O. This tuberculin is prepared by growing cultures of the tubercle bacillus on glycerine-beef-broth for 6 to 8 weeks, sterilizing the cultures by heat, filtering out the dead bodies of the bacilli, and finally evaporating the filtered fluid to one-tenth its original volume. For diagnostic purposes this tuberculin is employed in several ways. 1. On the skin. (a) Moro's test. An ointment (Moro's ointment) is prepared by mixing equal parts by weight of tuberculin and lanolin. Fifteen grains (1.0 gm.) of such an ointment are rubbed vigorously into the skin of the abdomen over an area of 2 or 3 inches in diameter. As a control an equal quantity of lanolin alone may be rubbed into a corresponding area. A positive reaction consists in the development upon the first area of a papulo-erythematous eruption in from one to three days. The number of papules varies from one to several hundred, and the activity of the reaction may be estimated by their number.

(b) Von Pirquet's reaction. For this purpose a 10 per cent. dilution of tuberculin is employed. Two minute scarifications or linear scratches are made upon the forearm, at a distance of 2 or 3 inches from each other. Into one of these a drop or two of the diluted tuberculin is rubbed. The other area serves as a control. In the tuberculous individual the test area becomes reddened, raised, and surrounded by an erythematous zone of varying size. The area is distinctly indurated and vesicles may form upon it. The reaction fades gradually in the course of several days and may persist for a week or more.

2. In the conjunctiva. Calmette's ophthalmic reaction. For this purpose a ½ to 1 per cent. solution of the dried alcoholic precipitate of old tuberculin is used. One or two drops of this solution are dropped in the conjunctival sac. Within twenty-four hours an active conjunctivitis develops in positive cases. The conjunctiva becomes swollen, reddened, and a watery, fibrinous or even purulent exudate appears, accompanied by lachrymation. The reaction has been so severe in many cases, resulting in permanent damage to the eye, that it is no longer recommended.

3. Under the skin. The subcutaneous injection of tuberculin is applicable only in afebrile cases. The dosage employed varies considerably in different hands. One milligram is a safe initial dose. If no reaction follows, a second injection of 2 milligrams may be given after two or three days. In case of failure after this dose larger doses may be tried, even up to 10 milligrams, but the smaller doses are usually satisfactory. To the subcutaneous injection of tuberculin the tuberculous patient reacts by a rise of temperature of several degrees, usually accompanied by more or less pain in the back and limbs and general malaise. A slight intensification of the physical signs in the chest may be noted. The reaction rarely lasts more than twenty-four hours. The subcutaneous injection of tuberculin should not be resorted to in very debilitated patients, nor in those with advanced disease of the heart, arteries, or kidneys.

Reaction to any of the tuberculin tests is evidence that the person tested has a tubercular lesion in some part of his body, but not necessarily an active process. Positive reactions are, therefore, frequently obtained in adults suffering from other affections. In childhood such reactions are much less common.

II. RÖNTGEN RAYS.—THE FLUOROSCOPE.—The screen enables one to study differences in the excursion of the diaphragm on the two sides due to impaired function of one lung. Inequality in the excursion is known as Williams' sign.

T h e S k i a g r a p h. Radiographic plates of the lungs, when well taken, give invaluable information as to the presence or absence of tuberculosis. Undoubtedly lesions may, in some cases, be demonstrated by the X-rays before they are appreciable by ordinary methods of examination. Difficulties arise from the fact that latent tuberculosis or other lesions may be misinterpreted. Expert training is necessary for correct interpretation of the plates (see Fig. 79).

Prognosis.—The prognosis is often very uncertain and difficult. Most cases of chronic phthisis eventually prove fatal, but that many cases result in what is practically a cure is shown by the great frequency with which healed or latent tuberculosis is found in the lungs of old persons who have died from some other cause. The disease may progress steadily and rapidly and be fatal in from six months to a year, or it may remain stationary for long periods and last for ten or even twenty years. It tends to progress more slowly in those of middle age than in young

persons. High fever, progressive emaciation, profuse expectoration, signs of extensive consolidation and softening, severe hemoptyses, laryngeal symptoms and persistent diarrhea are the chief unfavorable features.

FIBROID PHTHISIS

The term fibroid phthisis has sometimes been used to include all fibroid disease of the lungs whether tuberculous or not; but it is more properly restricted to that form of pulmonary tuberculosis in which

Fig. 79.—Extensive tuberculosis of the right upper lobe, which appears very dark as compared with the normal left apex.

fibrosis is the predominant change and caseation and softening are slight or altogether lacking. It is probable that in some of the cases the primary process has been a simple fibrosis upon which tuberculosis has become engrafted. The condition is not common and is met with chiefly in adults.

Morbid Anatomy.—As a rule the process begins at one apex and the lesion is usually confined chiefly to one lung. The lung is small, inelastic, tough, and shows throughout a great increase in fibrous tissue diffusely scattered or in dense bands. The pleura is firmly adherent and is often enormously thickened. Old caseous nodules may be found at

the apices or in the bronchial glands. The bronchi show a chronic bronchitis and often also bronchiectasic cavities. In the unaffected lung there is compensatory emphysema.

THE CLINICAL COURSE is extremely chronic. Cough is usually the chief symptom and is especially troublesome in the morning. Expectoration is profuse and results from the chronic bronchitis or from bronchiectasis. It may be fetid. Tubercle bacilli are scanty and may be absent for long periods. Fever is uncommon. There may be hemoptysis or pleuritic pain. The right heart eventually dilates and weakens and adds its special train of symptoms, such as dyspnea, cyanosis, ascites, etc.

THE PHYSICAL SIGNS are those of other types of chronic interstitial pneumonia: marked sinking and retraction of the affected side of the chest with drooping of the shoulder; feeble or absent expansion upon breathing; displacement of the heart; impaired resonance or dulness; feeble or tubular breathing, and moist râles or rhonchi. Bronchiectasis may give the signs of a cavity. The tuberculous nature of the process may be very difficult to demonstrate since tubercle bacilli are often absent from the sputum. The condition lasts for many years and may become serious only with the advent of cardiac weakness.

Prophylaxis.—Since the infectious matter is spread almost wholly by means of sputum the greatest care should be taken in properly disposing of this. Patients should be instructed scrupulously to avoid spitting on the floors of rooms, public buildings or conveyances, or even in the street. All sputum should be collected in spit-cups or wide-mouthed flasks containing a 5 per cent. solution of carbolic acid, or corrosive sublimate (1–500), or else should be received in pieces of cheesecloth or rags which can then be burned. If handkerchiefs are used the sputum should not be allowed to dry upon them and they should be thoroughly disinfected by boiling for one hour. This applies also to night gowns, bed linen and other articles which may be contaminated. If such precautions are faithfully observed the risk of infection to other members of the family is very slight indeed. The danger to young children from the use of milk from tuberculous cows is a real one and can be removed only by the careful and systematic inspection of dairies and cattle and the elimination of all animals suffering from the disease.

The children in tuberculous families should be reared with great care, especial attention being given to the avoidance of catarrhal troubles of the upper air passages, to the removal of adenoid growths and enlarged tonsils, and to the maintenance of vigorous health and good digestion. They should live an out-of-door life, if possible, in a mild, suitable climate, and their diet should include an abundance of milk, eggs and fats. Tuberculous mothers should not nurse their infants. Persons suffering from pulmonary tuberculosis, in an active stage at least, should not marry.

Treatment of Pulmonary Tuberculosis.—It is important to bear in mind that, in many cases, at least, of tuberculosis, there is a distinct tendency toward spontaneous cure, as is shown by the frequency with

which healed tuberculosis is found at autopsy. This tendency seems related to the state of general nutrition and the vigor of the vital processes rather than to local conditions in the lungs.

In like manner modern treatment is directed toward the building up of the general health and nutrition rather than to the lungs themselves.

TREATMENT OF THE DISEASE.—(a) *Climate and Fresh Air.*—An abundance of fresh air is the most important single remedy in the treatment of tuberculosis. Its importance, as distinguished from that of climate itself, has been much more generally appreciated in recent years, and this has led to most gratifying results. The fact that the patient must be treated at home and in the city should not prevent the systematic and careful carrying out of this measure. The plan is simply to give the patient, both in winter and summer, the greatest possible amount of fresh air and sunshine. In the daytime, if not bedridden, he should spend all of his time out of doors, properly protected from cold and wind. If confined to bed the windows should be widely opened and the bed kept in the sunlight as much as possible. Whenever possible, he should be moved to a couch on a sunny balcony or porch, well wrapped up and sheltered from the wind. At night the patient should occupy a room by himself, with the windows wide open. Cough, fever, sweating, etc., are not contra-indications to such open-air treatment. This plan of treatment is much more easily carried out in a suitably arranged sanitorium where the patients are constantly under medical supervision and discipline. In Europe many such sanitoria are to be found, and in this country they are, fortunately, increasing in number. The beneficial effects of sanitorium treatment are due quite as much to the systematic open-air life, and the intelligent regulation of diet, clothing, rest, exercise, etc., as to the effect of the climate itself.

Experience has shown that many varieties of climate may be suitable for consumptives; the essential features of all such being pure air (*i.e.,* freedom from dust), an equable temperature and an abundance of sunshine, so that the patient may be out of doors almost constantly. These requirements are usually best met in high, dry regions, but altitude in itself does not seem to be of primary importance. The high tablelands of Colorado, Arizona and New Mexico, and the regions about Davos and St. Moritz in Switzerland, are well-known instances of a dry, elevated climate. Many cases do extremely well in the cold, moderately elevated climate of the Adirondacks and the Catskills. If a warmer winter climate is desired it may be found in the comparatively dry regions of North Carolina, Georgia, Southern California, Algiers or Egypt, or in the moister climate of Florida or the Madeira or Canary Islands.

As to the locality best suited for an individual case only a few general rules can be given. The climate must first of all be one in which the patient can be constantly out of doors. The colder and more elevated regions are, therefore, best suited to the young and comparatively robust, and to those who are relatively free from fever and in whom the disease

is neither very active nor far advanced. Older patients, those with pronounced emphysema or arteriosclerosis; and those with advanced lesions, emaciation and continuous fever, usually thrive best in a warm and less elevated climate. Good food and proper nursing and care are quite as essential as a suitable climate and the good effects of climate may be altogether nullified if the former be lacking.

(b) *Diet.*—A gain in weight is one of the most trustworthy evidences of improvement, and the feeding should be regulated with that aim in view. Patients should be given just as much simple, wholesome food as they can eat and digest, even though, as is frequently the case, they have little or no appetite. The diet should be especially rich in proteids and fats in the form of milk, eggs, meat, cream, butter, etc. In many cases the dyspeptic symptoms are prominent and troublesome and only the simplest foods can be tolerated. In such cases milk, koumiss, broths, beef juice, beef jelly and the various prepared foods are suitable and should be given in small and frequent feedings. The anorexia should be combated by having the meals as appetizing and as daintily served as possible and by the use of bitter tonics, etc. When there is great repugnance to food or when, from involvement of the larynx, swallowing is difficult and painful, it may be necessary to feed by means of the stomach tube. Moderate fever is not a contra-indication to solid food unless the digestion be seriously disturbed.

(c) *Hygiene.*—The importance of an open-air life has been mentioned. The patient's sleeping room should be large, sunny and well ventilated; his clothing should be warm but light; the underclothing of wool but not heavy. Most phthisical patients, especially among the lower classes, wear much too heavy underwear and thereby increase rather than diminish the risks of taking cold. Thick flannel '' chest protectors '' are likely to do harm rather than good. The skin should be kept active and healthy by daily baths and rubbings.

Rest and exercise should be carefully regulated and systematized to suit the individual case and the latter should always stop short of real fatigue. Walking is usually the most suitable form of exercise. Moderate and simple arm and chest exercises frequently repeated are of decided value.

(d) *Drugs.*—There are no specifics for tuberculosis. Most of the drugs used act by improving the general nutrition and so increasing the powers of resistance.

Cod-liver oil is often of much value, especially in children. It should be given in small doses (51–2) one hour after eating. It is sometimes not well borne, and should not be continued if it disturbs digestion. Olive oil or cream is a useful substitute. Creosote, which has had a wide popularity in recent years, seems to act chiefly by lessening the cough and expectoration. Because of its disagreeable taste and its tendency to irritate the stomach it should be given after meals in capsules or disguised in an aromatic tincture or in whiskey. The beginning dose of one or two minims should be gradually increased to ten

minims three times a day, unless gastric disturbance is noticed. Creosote carbonate has a similar effect and is less irritating. It should be given in somewhat larger doses.

Arsenic, iron, malt extract and the hypophosphites are all useful tonics.

(e) *Specific Treatment.*—The use of the tuberculin of Koch and of its more recent substitutes is reserved for patients without fever and in good general condition. A variety of preparations of the tubercle bacillus are now used by different authorities in the treatment of tuberculosis. Of these the chief are (1) Koch's original tuberculin (O.T.), a filtered glycerine extract of cultures of the bacillus, heated to destroy any living bacilli. (2) T.R., tuberculin residuum, obtained by mechanically crushing or dividing cultures of the bacillus, treating them with salt solution, centrifuging and employing the precipitate or residuum. (3) B.E., an emulsion or vaccine prepared from cultures of the bacillus, and heated to 60° C. (4) B.F., bouillon fittré, is prepared by filtering bouillon cultures of the bacillus. The liquid is not heated. The dosage and method of using these preparations vary greatly. The principle is that of employing a minimum dose at the outset, so as to avoid any general reaction, repeating the injection at intervals of several days (4 to 5 or in some systems 10 to 12), and rapidly increasing the dose up to the limit of tolerance. The initial dosage of O.T. varies from 0.00000001 to as much as 0.0001 gram, and the maximum from 0.1 mg. to 1 gram. For the details of these methods special articles such as that in Osler's Modern Medicine, Vol. III, p. 415, must be consulted.

TREATMENT OF SPECIAL SYMPTOMS.—*Cough.*—The measures directed toward the relief of the disease itself are usually effective in lessening the cough. The routine administration of cough syrups should be avoided if possible, as they are apt to disturb digestion. The cough is frequently due to the complicating laryngitis and may be relieved by appropriate local treatment. Inhalations of creosote, turpentine, menthol, etc., are sometimes efficacious. Frequently, however, some such sedative as codeine (gr. $\frac{1}{8}$–$\frac{1}{4}$) or heroin (gr. $\frac{1}{12}$–$\frac{1}{8}$) or morphine (gr. $\frac{1}{12}$–$\frac{1}{8}$) is needed. At other times some stimulant expectorant may act best. If cavities are present a certain amount of coughing is necessary for their proper evacuation.

Temperature.—Fever, unless high, requires no special treatment and should not be allowed to interfere with the fresh air régime. As a rule, patients with fever must be kept at rest, either in bed or upon a couch or reclining chair. If persistently high the fever may be controlled somewhat by frequent cool spongings or by small doses of antifebrin or phenacetin.

Night-sweats may often be controlled by giving an alcohol sponge bath at bedtime and by keeping the sleeping-room cool and the covering light. If drugs are needed, atropin (gr. $\frac{1}{120}$–$\frac{1}{80}$) is the most satisfactory. Other drugs used are aromatic sulphur acid (m. xx), agaricin (gr. $\frac{1}{10}$–$\frac{1}{4}$), picrotoxin (gr. $\frac{1}{60}$) and camphoric acid (gr. xx).

Hemoptysis.—Absolute quiet in bed and morphine subcutaneously to control the coughing are the measures of chief importance. An ice bag may be placed over the affected region of the chest. Thrombus formation is favored by a lowering of the general blood pressure and this is best accomplished by a low diet, by purging and by repeated small doses of aconite or nitroglycerin. The use of ergot, adrenalin, tannic acid, etc., is irrational and should be avoided.

Pleuritic PAIN, which is sometimes very troublesome, is best treated by some form of counter-irritation, such as tincture of iodine, mustard pastes or the actual cautery.

V. TUBERCULOSIS OF THE CIRCULATORY SYSTEM

Tuberculosis of the heart and blood-vessels is rare at best, and is probably never primary. In the myocardium a few scattered miliary tubercles are occasionally seen, and, very rarely, also larger caseous areas. A few cases of tuberculous endocarditis have been recorded. Tuberculosis of the aorta and the larger arteries is extraordinarily rare. On the other hand, the extension of a tuberculous process in the lungs or lymph-nodes to the walls of adjacent small veins or arteries is not uncommon, and this seems to be the usual way in which distribution of the bacilli occurs. Tuberculosis of the pericardium is described with that of the other serous membranes.

VI. TUBERCULOSIS OF THE LYMPH–GLANDS

(Scrofula)

Positive proof of the unity of scrofula and tuberculosis was lacking until the demonstration by Koch of the tubercle bacillus in both. The lymph-nodes show an especial susceptibility to the action of the bacilli and in children scrofula is much the commonest form of tuberculosis. Even in adults the lymph-nodes are frequently invaded and the condition is sometimes met with in advanced life. In children the disease is most common in those living in the dark, crowded and poorly ventilated homes of the poor and in asylums. Negroes seem especially prone to this form of tuberculosis. The frequence of bovine bacilli is notable (see p. 387).

Morbid Anatomy.—The anatomical changes are similar to those found in other forms of tuberculosis; miliary tubercles, caseation, softening, sclerosis, and calcification occurring in varying combinations and proportions. Usually the process is a slow and relatively benign one.

Clinical Types.—Several types are distinguished, depending upon the particular group of nodes involved:

(a) CERVICAL.—This is a very common type in children. Infection occurs in most cases through the mouth, nose or pharynx. The tonsils, especially, are now regarded as important portals of entry. The passage of the bacilli through the mucous membrane is doubtless much favored by catarrhal inflammation or other changes.

One after another of the cervical nodes becomes enlarged until the whole chain on both sides of the neck may be involved. The process is usually very slow and the nodes remain for a long time firm, painless and separate. Eventually in most cases, they fuse into large masses and not infrequently undergo suppuration. When this happens the skin overlying the mass becomes red and adherent and the abscess may rupture externally.

(b) Bronchial.—The lymph-nodes at the roots of the lungs are constantly involved in pulmonary tuberculosis. They may also become infected by extension of the process from the cervical nodes and also, it is believed, by the passage of bacilli through the bronchial mucous membrane even when no pulmonary tuberculosis exists. In some cases infection of the lungs seems to have been secondary to the tuberculous changes in these nodes. In children tuberculosis of the bronchial lymph-nodes is the most constant and not infrequently the only lesion found at autopsy. It appears to be the primary lesion in the great majority of the cases of tubercular infection in children in this country and from it many of the cases of acute general miliary tuberculosis originate. Although the nodes often form large tumor masses serious compression of the neighboring structures is uncommon. Rarely such caseous and softened glands have been known to perforate a bronchus, the trachea, the esophagus, the pulmonary artery and even the aorta.

(c) Mesenteric (Tabes mesenterica).—Tuberculosis of the mesenteric and retroperitoneal nodes is especially frequent in young children. Infection occurs regularly through the intestines, in many cases by means of contaminated milk. The intestine may be, but frequently is not, itself the seat of tuberculous lesions. The condition is not uncommon in pulmonary tuberculosis and is then usually due to the swallowing of sputum. The diseased nodes often form large tumor masses which may sometimes be felt through the abdominal wall. In children the symptoms are chiefly those of progressive emaciation and anemia. The abdomen is distended and there is usually persistent diarrhea. In many of the cases there is an associated tuberculous peritonitis. The disease is usually fatal.

(d) General Tuberculous Adenitis.—An uncommon form of tuberculous adenitis is that in which there is a general enlargement of the lymph-nodes all over the body. It is usually associated with moderate fever and with progressive wasting. The condition may closely simulate Hodgkin's disease.

VII. TUBERCULOSIS OF THE SEROUS MEMBRANES

Tuberculosis of the Pleura.—Tuberculous pleurisy is commonly secondary to tuberculosis of the lungs or of the bronchial nodes. It may appear in connection with tuberculosis of other serous membranes, *e.g.*, the pericardium and peritoneum, or it may be a primary condition. It has come to be realized in recent years that many, if not most, of the cases of so-called idiopathic or simple pleurisy are really tuberculous in nature.

The disease appears in acute, subacute and chronic forms, and the character of the exudate may be fibrinous, serous, hemorrhagic or purulent. Frequently the serous effusion has a distinct greenish tinge. An occasional and very chronic form is that associated with the active proliferation of new connective tissue; adhesion of the two layers, and the conversion of the pleura into a dense mass of fibrous tissue which may be an inch or more in thickness.

The symptoms and physical signs of tuberculous pleurisy do not differ from those of other forms of pleurisy. (See pages 42–56.)

Diagnosis.—Positive evidence of the tuberculous nature of a given pleurisy is sometimes very difficult to obtain. If tuberculosis can be demonstrated in the lungs or elsewhere there is little doubt as to the nature of the pleurisy. If the effusion be hemorrhagic the probability is that the inflammation is either tuberculous or cancerous. Acute idiopathic serous pleurisy is tuberculous in the majority of cases. Tubercle bacilli are present in the exudate (either serous or purulent) only in very small numbers and are difficult to find by the ordinary procedures. The tuberculous nature of a serous effusion can often be proven by the injection of several cubic centimetres into the peritoneum of a guinea pig. A marked preponderance of lymphocytes in the cells of the exudate is suggestive of tuberculosis. The von Pirquet reaction is also of some value, and the patients may, if necessary, be tested by tuberculin injections after the fever has subsided. (See page 47.)

Tuberculosis of the Pericardium.—This somewhat rare disease is usually secondary to tuberculosis of the lungs, or the bronchial and mediastinal lymph-nodes, or is associated with tuberculosis of the other serous membranes. The lesion may be that of an acute or subacute inflammation with a predominance of fibrinous or of serous exudate, or may be a chronic proliferative process with adhesions of the two layers and complete or partial obliteration of the pericardial sac.

The signs and symptoms do not differ from those of non-tuberculous types of pericarditis.

Tuberculosis of the Peritoneum.—ETIOLOGY.—The peritoneum is regularly involved in acute general miliary tuberculosis and in that form of tuberculosis which attacks several of the serous membranes. Usually tuberculosis of the peritoneum is secondary to that of the lungs, intestines, mesenteric nodes, Fallopian tubes, bladder or epididymis. The disease is most common in children and young adults, but may be met with at any age. It is more frequent in females than in males. Cirrhosis of the liver, ovarian cysts and hernia seem distinctly to predispose to the disease.

Morbid Anatomy.—Both the lesions and symptoms present many variations. There may be an acute and extensive eruption of miliary tubercles over the parietal and visceral peritoneum; and associated with this an exudative inflammation with the formation of a varying amount of serofibrinous or hemorrhagic exudate. In its more chronic form the disease presents two types of lesions. In one there is the develop-

ment of large tuberculous nodules which tend to caseate, soften, undergo suppuration and sometimes to perforate the intestines. Sacculated collections of pus are not uncommon. In other cases the chief change is a fibrous overgrowth, with great thickening and retraction of the omentum and mesentery, and sometimes with enormous thickening of the peritoneal coat of the intestines.

SYMPTOMS.—In the acute cases there are continued fever, abdominal pain and tenderness, muscular rigidity, and, often, ASCITES. The effusion is usually serous, but may be seropurulent or hemorrhagic. If ascites is lacking and tympanites and diarrhea present the symptoms may closely resemble typhoid fever. The chronic cases present a very variable clinical picture. Often the only symptoms are progressive loss of flesh, a slight, irregular temperature, and a retracted, rigid and somewhat tender abdomen. A subnormal temperature is not uncommon. In many cases one or more tumor masses can be felt. The masses may be formed of the retracted and thickened omentum, of matted intestines, of sacculated fluid, or, rarely, of greatly enlarged mesenteric nodes. The most characteristic of these masses is that made by the shrunken omentum, which often forms a somewhat cylindrical tumor lying across the upper part of the abdomen. Ascites may be present in either the acute or the chronic type.

DIAGNOSIS.—The acute cases without ascites often simulate typhoid. The spleen, however, is usually not enlarged, the typical eruption and the Widal reaction are lacking and there is usually more abdominal tenderness and rigidity. In the chronic cases the presence of one or more tumor masses may lead to the diagnosis of a malignant growth or an ovarian cyst. Afebrile cases with ascites may be difficult to distinguish from cirrhosis of the liver. In all cases careful search should be made for evidences of tuberculosis in other parts of the body. The patient may be tested by tuberculin either on the skin (von Pirquet) or by injection (see p. 402).

PROGNOSIS.—Although the prognosis is, in general, bad, a considerable proportion of cases recover either spontaneously or as a result of operation. Cases with purulent exudation are usually fatal. The duration of the disease varies from months to years.

TREATMENT.—Every case should be given a thorough trial of treatment by such general measures as are recommended in the treatment of pulmonary tuberculosis. If no benefit is derived from these, laparotomy should be performed. Just why the simple opening of the abdomen and evacuation of the fluid should be so often followed by improvement or cure is still obscure, but the fact cannot be denied. Aspiration of the fluid alone has but little effect.

General Serous Membrane Tuberculosis.—PICK'S DISEASE— CHRONIC POLYSEROSITIS.—It happens occasionally that several serous membranes may be simultaneously affected, even when there is no evidence of a general dissemination of tubercles or of marked visceral lesions. The peritoneum and the pleura are the membranes most fre-

quently affected, but the pericardium also may be involved. The lesions and symptoms do not differ from those already described under the separate forms of serous membrane tuberculosis.

VIII. TUBERCULOSIS OF THE GENITO-URINARY SYSTEM

The genito-urinary tract in both sexes is not infrequently the seat of tuberculous disease; the kidneys, Fallopian tubes and testicles being the parts most commonly affected.

Tuberculosis of the Kidney.—ETIOLOGY.—The kidneys are frequently involved as a part of general miliary tuberculosis, but, as a rule, give no distinctive symptoms. Local tuberculosis of the kidney may be primary, but is usually secondary to some focus elsewhere in the body, the infection occurring by means of the blood. Infection may, however, be by direct extension from the peritoneum, from carious vertebræ, or from the bladder and ureter. The disease is much commoner in the male sex and is most often seen in young adult life.

MORBID ANATOMY.—The disease may be, but usually is not, confined to one kidney. The process seems usually to begin in the pyramids or calyces. Tuberculous nodules develop, undergo caseation and suppuration, and gradually invade the kidney substance so that eventually the organ may become riddled with cavities containing caseous material or pus. The pelvis also becomes involved in many cases and the ureters and bladder may be secondarily infected.

SYMPTOMS.—These often extend over many months or even years and show much variation in severity. There may be lumbar pain and tenderness, slight or irregular fever, loss of flesh, chills, sweats, etc. Frequent and urgent micturition may occur even without disease of the bladder. The urine usually contains pus and albumin and sometimes blood or fragments of cheesy matter. Careful examination will often reveal tubercle bacilli. The kidney may be of normal size or may show moderate enlargement.

The diagnosis from other forms of pyelitis is sometimes very difficult. In the absence of evidence of tuberculosis elsewhere in the body the positive diagnosis can be made only by the finding of tubercle bacilli in the urine or by the positive response to the tuberculin test.

TREATMENT.—If the disease be confined to one kidney and if the bladder be not involved the kidney should be excised. Under other circumstances the condition can be treated only along the general lines indicated in all forms of tuberculosis.

Tuberculosis of the Bladder.—Involvement of the bladder is usually secondary to disease of the kidney or of the testis and prostate. The tuberculous nodules soon break down and form ulcers of varying extent, which are most common in the region of the trigone. The symptoms are usually those of a subacute or chronic cystitis, but may simulate those of a calculus.

Tuberculosis of the Testicle.—This may be primary or secondary. Usually but one organ is involved. The disease is seen in young children

as well as in adults. The process affects chiefly the epididymis, but the testis proper may be invaded, and the disease in time extends along the vas deferens to the seminal vesicles and prostate.

Tuberculosis of the Fallopian Tubes and Uterus.—Disease of the tubes is much commoner than that of the uterus. The process is usually secondary to tuberculous lesions elsewhere and infection may occur by direct extension as from the peritoneum or through the blood. Moreover, the possibility of direct infection from the male should be borne in mind.

IX. TUBERCULOSIS OF THE NERVOUS SYSTEM

Acute tuberculous meningitis is described elsewhere (page 523). Aside from this form, tuberculosis occurs in the brain and cord as a chronic localized meningitis and in the form of tuberculous tumors known as solitary tubercles. These solitary tubercles are met with chiefly before the age of fifteen. They may be single or multiple, and consist of a firm, rounded, circumscribed caseous mass, sometimes several centimeters in diameter. They are found in the cerebellum, cerebrum, pons or spinal cord and do not differ in their symptoms from other varieties of tumors. They are usually secondary to tuberculosis in other parts of the body. The solitary tubercle is the most frequent type of brain tumor in children. (See page 545.)

LEPROSY

Leprosy is a chronic infectious disease caused by the BACILLUS LEPRÆ and marked by macular skin eruptions and by nodular growths in the skin, mucous membranes and nerve tissues.

Etiology.—The disease has been known for very many centuries. At the present time it prevails in China, India, Africa, Norway, in parts of Russia, and in the Hawaiian Islands. In this country the disease exists in Louisiana, in Minnesota among the Norwegians, and on the Pacific coast among the Chinese.

The BACILLUS LEPRÆ, discovered by Hansen in 1871, closely resembles the tubercle bacillus both in morphology and in its staining qualities. It is found in great numbers in the specific nodules and in the discharges from the nose, throat and the skin ulcers, and in the blood. For many years efforts to cultivate the organism artificially failed, but it has recently been grown together with amebæ and symbiotic bacteria and Duval has obtained pure cultures in media containing tryptophan and other free amido-acids. Inoculations of pure cultures have been made in animals, and in monkeys a disease closely resembling human leprosy has been produced.

The disease tends to run in families, but is probably never directly inherited. It may occur at any age beyond infancy. Long-continued and close contact with the disease is usually required for infection, but it may occur by means of infected clothing or other fomites. The secretion from the nose and throat seems to be the chief means of dissemination of the contagium. Overcrowding, filth and uncleanly habits are

strong predisposing influences. Hutchinson believes that infection occurs through the eating of uncooked fish.

Morbid Anatomy.—The specific lesion of leprosy is a granuloma similar to the tubercle of tuberculosis and the gumma of syphilis. It occurs in the form of distinct nodules or tubercles and of diffuse infiltrations. These growths are composed of small cells and a connective-tissue stroma and contain vast numbers of bacilli. They occur chiefly in the skin and nerve-trunks, but are also found in the mucous membranes and in such viscera as the testicles, liver and spleen. These growths tend often to break down and form ulcers.

Symptoms.—The period of incubation is usually two or three years in length. Before characteristic symptoms appear there are often for weeks or months vague prodromata such as fever, headache, prostration and fleeting pains. The first symptom is regularly the appearance in the skin of the face, trunk and extremities of hyperesthetic erythematous MACULES of varying size, which often become pigmented, but may lose their color and grow white and anesthetic.

Sooner or later the characteristic granulomata develop. When these occur chiefly in the skin the type is called tubercular or nodular leprosy, when in the nerve-trunks it is known as anesthetic or nerve leprosy.

TUBERCULAR LEPROSY.—Purplish swellings appear in the skin of the face, trunk and extremities, which gradually grow into distinct nodules or diffuse thickenings. The face is usually the chief seat of these growths and is often greatly deformed and thickened ('' leontiasis ''). Similar growths appear in the mucous membrane of the conjunctivæ, nose, larynx, etc. Ulceration may cause much destruction of tissue or result in deforming scars. Fingers and toes are often destroyed and nodules may cause enlargement of the testicle or liver. Ulceration of the larynx is common. The patient eventually dies from exhaustion, sepsis or some complication.

ANESTHETIC LEPROSY.—The symptoms after the appearance of the macules are chiefly those of a slowly developing neuritis affecting the extremities—pain, hyperesthesia, anesthesia, paralysis, contractures, trophic disturbances, bullæ, gangrene, etc. Along the course of such superficial nerve-trunks as the ulnar the nodular swellings can often be felt.

Not infrequently the lesions affect both the skin and nerve-trunks and the symptoms of the two types are combined.

Diagnosis.—In well-developed cases the diagnosis is readily made. In the early, macular stage, the presence of hyperesthesia or anesthesia in the erythematous patches is significant. The bacilli are readily found in the discharges of ulcers and in the nodules, or in the blood.

Prognosis.—Most cases eventually prove fatal. The average duration of life in the tubercular type is eight or ten years. In the anesthetic type it is often twenty or thirty years.

Treatment.—Some form of segregation is necessary. Attention should be given also to the proper disposal of the infectious material and

to the disinfection of fomites. No specific remedy is known. Chaulmugra oil is the drug most esteemed and is given in increasing doses up to 40 minims three times a day. Many other remedies have been used, including ichthyol, potassium iodide, bichloride of mercury, arsenic, etc. It is important that the patient be put under the most favorable hygienic conditions and that everything possible be done to improve his general health and increase his powers of resistance.

TETANUS

(Lock-Jaw)

Definition.—Tetanus is a very fatal infectious disorder, resulting from inoculation of a specific bacillus and marked by persistent and increasing spasm of the muscles of the jaw, neck and trunk.

Etiology.—In most cases the disease follows some wound of the body surface and especially of the hands or feet. Punctured and badly contused or lacerated wounds are those most likely to result in tetanus. Clean, incised wounds are rarely, if ever, infected. Toy pistol wounds seem especially dangerous. Many cases have developed in puerperal women, and also in new-born infants (from umbilical infection). Occasionally no wound is discoverable and the cause has been ascribed to exposure to cold. Tetanus occurs endemically in various hot and temperate countries and is seen at all ages.

The BACILLUS OF TETANUS is a motile, spore-bearing anaërobic organism, found often in the soil, in manure, in street dirt, etc., and it is the wounds contaminated by such dirt that are most liable to tetanus infection. The anaërobic character of the germ explains why it thrives best in deep, punctured wounds. The bacilli multiply in and about the wound, but are found nowhere else in the body. They produce an extraordinarily virulent toxin, to whose action upon the motor nerve-centers all the symptoms of the disease are due. The toxin seems to reach these centers rather by travelling along the axis-cylinders of the peripheral nerves than by means of the circulation.

Morbid Anatomy.—No constant lesions are found after death. The brain and cord often show hyperemia, perivascular exudation, and sometimes minute hemorrhages.

Symptoms.—The INCUBATION PERIOD varies from a few days to three weeks. In general, the shorter the incubation the more severe are the symptoms. These begin with slight stiffness and soreness of the muscles of the neck and jaw. The stiffness rapidly increases until the jaws cannot be opened (trismus, lock-jaw). Spasm of the other facial muscles may produce a curious fixed grin (RISUS SARDONICUS). The rigidity extends to the back, chest and abdomen, but the arms and legs usually escape. Soon the rigidity is increased by sudden tonic spasms of the affected muscles. These last but a fraction of a minute, but are usually intensely painful. In them the back may be arched so that the body rests only on the head and heels (opisthotonos). These

tonic spasms become more and more frequent and severe and are induced by any slight external irritation, such as a noise or touch. Between the spasms there always remains some rigidity. The temperature, as a rule, is normal or nearly so until late in the disease, when it gradually rises to 103°–105° F. The mind is usually clear throughout. The spasms finally become almost continuous and the patient dies from exhaustion or from sudden failure of heart or respiration.

The DURATION is usually from three to seven days. MILD cases are seen which run a more protracted course and end in recovery. A rare form—the head-tetanus of Rose—is that which follows wounds of the face and is characterized by paralysis of the face on the side of the wound, trismus and great difficulty in swallowing. Such cases may resemble hydrophobia.

Diagnosis.—Confusion can hardly occur if the history of the case and the progression of the symptoms be carefully considered. The bacilli can sometimes be found in the pus of the wounds.

Trismus alone may appear in hysteria and in such affections as quinsy and mumps. In TETANY the spasm affects chiefly the extremities. In STRYCHNINE POISONING the onset is abrupt and violent, the rigidity is not constant, and trismus is uncommon.

Prognosis.—Death results in quite three-fourths of the cases. A short period of incubation, early fever and the rapid development of rigidity are unfavorable signs.

Treatment.—The wound should be excised or be freely opened, cauterized and treated antiseptically. To lessen the violence of the spasmodic paroxysms full doses of chloral or morphine or inhalations of chloroform should be used. It may be necessary to feed the patient by a catheter passed through the nose.

An ANTITOXIN has been developed which in animals has been shown to neutralize completely the effects of the tetanus toxin, but which clinically has proved somewhat disappointing; probably because it can rarely be given before the disease is well developed. Some good results have been obtained from injecting the antitoxin directly into the lateral ventricles of the brain, and very recently a few brilliant cures have resulted from its injection into the main nerve-trunks leading from the infected region.

Antitoxin has also been used prophylactically, especially after Fourth-of-July wounds, with excellent results.

FEBRICULA

(Ephemeral Fever)

Febricula is not a specific disease, but is merely a term applied to a not uncommon type of febrile attack, lasting from one to ten days, in which there are no localizing symptoms or signs and in which no cause can be ascertained. Such attacks are frequent in children and may be

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met with at any age. Many of these cases, especially in children, are doubtless of gastro-intestinal origin—indigestion, mild gastro-enteritis, food poisoning, constipation, etc., in which fever may be almost the only prominent symptom. Other cases are very mild and atypical examples of such infectious diseases as typhoid, scarlet fever and influenza. Sometimes the cause may be a tonsillitis, pharyngitis, or bronchitis, unrecognized because of the slight local signs. Other possible causes are otitis media, rheumatic infection, sewer gas poisoning, etc.

Symptoms.—The fever is usually not high (100°–103° F.), nor is the pulse much disturbed. Headache and muscular pains are common. The tongue is coated, the appetite lost and the bowels often constipated. The fever may subside within two or three days or may last for a week or more. The patients regularly recover.

Diagnosis.—The term should be applied to a febrile attack only after repeated careful examinations have failed to disclose any of the above-mentioned conditions.

Treatment.—Rest in bed, a fluid diet, a calomel purge and a simple diaphoretic mixture are all that is needed in the way of treatment.

WEIL'S DISEASE

Definition.—Weil's disease is an acute febrile jaundice, probably of infectious nature, which sometimes appears in small epidemics. A bacillus of the proteus group has been found in the urine and tissues, but the true nature of the disease is still uncertain. It is seen chiefly in the summer months and in young adult males. Many of the cases have been in butchers.

The only lesions found POST-MORTEM have been fatty degeneration of the liver cells, degeneration of the renal epithelium and congestion of the viscera and gastro-intestinal mucosa.

Symptoms.—The disease begins abruptly with a chill, high fever, severe muscular pains and headache. Jaundice appears on the first or second day, is usually severe and is of the obstructive type, as shown by the light-colored stools. The fever lasts from one to two weeks, is usually remittent and falls by lysis. There may be delirium or stupor. The liver is swollen and tender, and the spleen much enlarged. The urine shows evidences of a mild nephritis. Vomiting and epigastric pain may be present. Most of the cases recover.

Diagnosis.—The epidemic character, the high fever and the jaundice, the swelling of the liver and spleen and the low mortality are the chief distinguishing features. Acute yellow atrophy, yellow fever and catarrhal jaundice are the conditions most likely to lead to confusion.

The TREATMENT is symptomatic.

MILIARY FEVER
(Sweating Sickness)

Miliary fever is an epidemic febrile affection occasionally seen in certain districts in France, Italy and Austria. The epidemics affect large numbers of people, but are usually of very short duration. The symptoms are those of fever, profuse sweating and, in the course of three or four days, an abundant eruption of miliary vesicles. Most of the cases recover within a few days, but the severe form of the disease is marked by grave toxemic symptoms and is often fatal.

GLANDULAR FEVER

Definition.—This is an acute infectious disease of children marked by fever and by inflammation and swelling of the cervical lymph-glands. The disease is usually seen in the form of house epidemics. It attacks children of all ages, but rarely occurs in adults. Infection is supposed to occur through the tonsils or the pharyngeal mucosa, but the exciting germ is yet unknown. Some of the cases may be of influenzal nature.

Symptoms.—The disease begins abruptly with pain and stiffness of the neck, fever, headache and often vomiting and abdominal pain. The pain and stiffness increase and on the second or third day the enlargement of the glands is noticed. This affects chiefly the anterior cervical glands, but may involve also the glands in other parts of the body. The glands vary in size from a bean to an egg, and are tender and painful. Only rarely, however, do they suppurate. The skin is usually not reddened, nor is there much periglandular swelling. There is usually enlargement of the spleen and liver. The tonsils and throat show no signs of acute inflammation. The acute symptoms subside in ten days or two weeks, but the glandular swellings disappear more gradually.

Suppuration of the glands, otitis media and hemorrhagic nephritis are occasional complications.

Diagnosis.—Other causes of glandular swelling, such as tonsillitis, diphtheria and tuberculosis, must be excluded and the epidemic character of the disease should be borne in mind.

The prognosis is good and the treatment palliative and symptomatic.

MILK SICKNESS

This is a curious disorder now rarely seen, but at one time prevalent and serious in certain of the new settlements west of the Alleghanies. It is associated with, and seems to depend upon, an infectious disease of the domestic animals (cattle, sheep and horses), called "the trembles." The affection is believed to be contracted by eating the meat or drinking the milk of diseased animals.

The symptoms, in man, begin after a few days of vague prodromata with severe abdominal pains, nausea and vomiting. There are also fever, thirst, obstinate constipation, a tremulous, swollen tongue and a very foul breath. In severe cases delirium, convulsions or coma may develop.

The disease may prove fatal within two or three days; but the symptoms usually last from one to three weeks and most of the patients recover.

ROCKY MOUNTAIN SPOTTED FEVER
(Tick Fever; Black Fever, or Blue Disease)

Definition.—An acute infectious disease, closely resembling typhus fever, met with in Montana, Idaho and adjacent parts of the Rocky Mountains.

Etiology.—The disease occurs only in mountainous regions and in the spring or summer months. It is transmitted by the bite of the wood tick, dermacentor occidentalis, and can be conveyed to monkeys, rabbits, or guinea pigs by injections of infected blood, but many careful studies have failed to identify a causative organism.

Morbid Anatomy.—A petechial rash on the skin, general hypostatic congestion, enlargement of the liver and spleen, with degenerative lesions in all the viscera are found. Cultures are negative.

Symptoms.—A fever, rising rapidly, often with chills, continuous with moderate remissions, subsiding after two weeks and reaching normal toward the end of the third; an eruption, beginning on wrists and ankles and spreading to the whole body, at first roseolar, later petechial; severe nervous symptoms, often ending in coma; enlargement of the liver and spleen; digestive disturbances proportionate to the fever, constitute the essentials of the disease.

The blood shows a leukocyte count of 8,000 to 14,000.

Diagnosis.—Typhoid fever can be excluded by the absence of the Widal reaction, the negative blood cultures, and no intestinal lesions, but at present no sure criteria distinguish it from typhus fever.

PELLAGRA

Definition.—An endemic disease, characterized by an erythema of exposed surfaces, marked emaciation, profound melancholia and sometimes mania.

Etiology.—Adults from 20 to 50 years, and men especially, are attacked. The disease is endemic throughout Southern Europe, particularly in Italy, in North Africa, South America, and in some parts of the Southern States. Field laborers suffer most frequently.

It has been attributed to many causes, from exposure to the sun, to syphilis, and most persistently to the eating of maize, but none of these theories is satisfactory.

The disease occurs year after year in the early spring months, continues through the summer, subsides during the fall and winter.

Morbid Anatomy.—Emaciation is extreme. The viscera show chronic degeneration. The brain and cord show chronic leptomeningitis with thickening, perivascular infiltration, and degeneration of the nerve-cells.

Symptoms.—A patchy erythema appears on the backs of the hands and feet, later on face, arms, legs, and trunk (see Fig. 80). Petechiæ

or blebs may form on the swollen areas. After a fortnight desquamation sets in and the skin becomes dry, rough and brownish. Burning or itching accompanies the eruption.

Weakness, sleeplessness or uncontrollable sleepiness and depression are common.

The tongue is coated, appetite lost, nausea or vomiting often present, and constipation the rule.

Two or three months after the onset the symptoms subside, only to recur the next spring. With each recurrence the patient becomes weaker, more emaciated and depressed. Periods of maniacal excitement with a tendency to suicide develop. Finally the patients become demented, paralytic, bed-ridden, and die of exhaustion, or from intercurrent disease.

Fig. 80.—Pellagrous eruption in stage of desquamation. (International Clinics, Vol. I, Ser. 21.)

The duration of pellagra varies from two years to ten or fifteen.

Diagnosis.—The combination with debility and marked nervous symptoms is characteristic.

Prognosis.—If taken early, the disease may be cured. Late cases are hopeless.

Treatment.—Removal from an infected district, good care and tonics, especially arsenic, are indicated.

GLANDERS

(Farcy)

Definition.—A very fatal specific infectious disease of the horse, communicable to man, which is marked by granulomatous lesions in the nasal and respiratory mucous membranes and beneath the skin.

Etiology.—The disease is common in horses, asses and mules. In man it occurs by inoculation through some break in the skin or mucous

membrane and is seen almost exclusively in stablemen and others who work about horses.

The exciting germ is a small, short, non-motile bacillus, BACILLUS MALLEI, which has peculiar staining qualities and is found abundantly in the specific lesions.

Morbid Anatomy.—The lesions consist of small granulomatous nodules, composed of lymphoid and epithelioid cells, which tend to suppurate and break down. They are found in the mucosa of the nose and air passages, where they form ulcers; along the course of the subcutaneous lymphatics, where they form abscesses, and in the internal organs. When the lesions occur chiefly in the mucous membranes the

FIG. 81.—The pustular eruption of glanders, often mistaken for small-pox.

condition is called GLANDERS; when they are chiefly beneath the skin it is known as FARCY.

Symptoms.—The incubation period varies from a few days to several weeks and is shortest in the acute and rapidly fatal cases. Both glanders and farcy occur in an acute and chronic form.

ACUTE GLANDERS runs its course in about a week, and is regularly fatal. It begins with fever, pains and prostration. The nasal mucosa is much swollen; the granulomata break down and cause ulceration and there is an offensive, purulent discharge. A papulo-pustular eruption appears over the face, and often over other portions of the body, which may simulate small-pox (see Fig. 81). The patient dies of the intense constitutional disturbance or from a complicating pneumonia.

CHRONIC GLANDERS shows itself as a chronic nasal and laryngeal catarrh with ulceration.

PLATE IV.
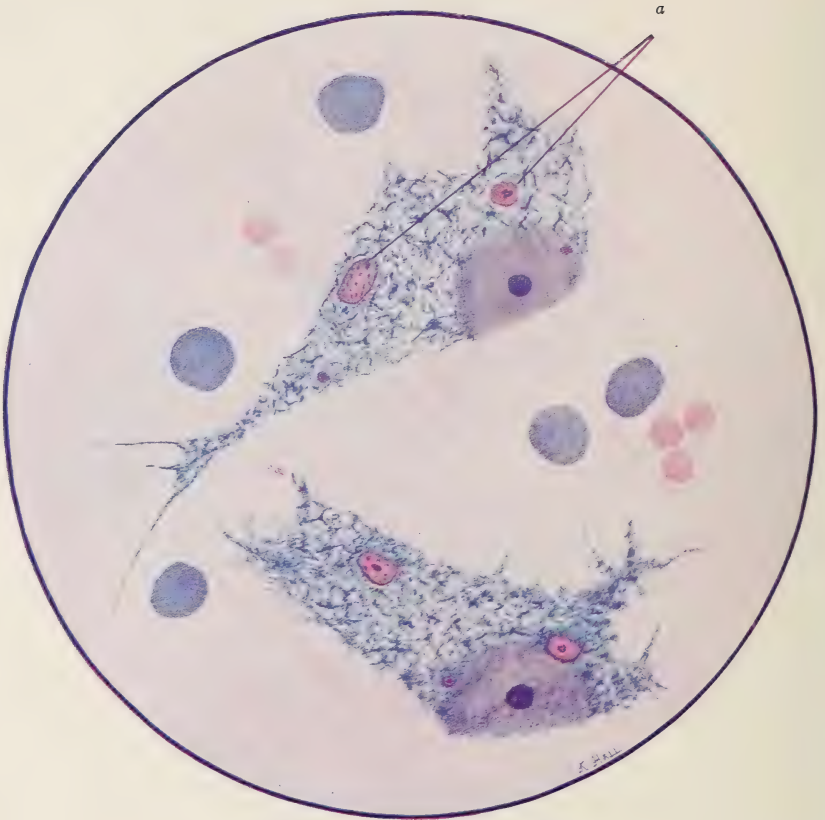
Negri bodies (*a*) in a smear preparation of the hippocampus major of a dog; stained with eosin and methylene blue. (From International Clinics, Vol. III, Series 20, page 151.)

ACUTE FARCY results from infection of the skin and appears as a very severe cellulitis with secondary abscesses (''farcy buds'') along the course of the subcutaneous lymphatics and elsewhere. The disease runs the course of a pyemia and is fatal, as a rule, within a week or two.

In CHRONIC FARCY there are sluggish subcutaneous nodules which eventually suppurate and form ulcers.

Diagnosis.—This depends upon the history of possible infection, the clinical picture described and the finding of the bacillus mallei in the abscesses. In animals mallein, a glycerine extract of a culture of the glanders bacillus, is used subcutaneously as a means of diagnosis. In the guinea pig, if pus containing the bacillus mallei be inoculated into the peritoneum, within two or three days, a characteristic orchitis is developed. This reaction is helpful in the identification of the organism.

Treatment.—The point of infection should be excised or thoroughly cauterized; the abscesses should be opened as they appear, and the patient's strength maintained by supportive measures.

HYDROPHOBIA

(Rabies)

Definition.—Hydrophobia is an acute infectious disease of the central nervous system occurring in certain of the lower animals and communicated to man by the saliva of such diseased animals.

Etiology.—Dogs, wolves and cats are the animals chiefly affected, but many other species are susceptible to the disease. Rabies is conveyed to man chiefly by the bites of mad dogs, and it is in countries where dogs are permitted to roam unmuzzled that most of the cases are seen. In Russia many cases occur from the bites of wolves. The disease is comparatively rare in this country.

Probably not more than one-fourth of those bitten by rabid dogs develop the disease. Bites upon the face, head and hands (the unprotected parts) are especially dangerous. The virus is found chiefly in the central nervous system and in the saliva. From the point of inoculation it seems to travel to the cerebrospinal axis along the nerves and not by means of the blood or lymph. The nature of the virus is obscure, but the disease probably depends upon some unknown microorganism.

Morbid Anatomy.—Lesions are found in the medulla, brain and spinal cord. There are degenerative changes in the ganglion cells and nerve-fibers, as well as congestion of and exudation from the blood-vessels. The mucous membranes of the respiratory and digestive tract are much congested. In the cells of the central nervous system, especially those of the cornu ammonis and the cerebellum, certain minute bodies, the Negri bodies, staining readily by eosin and methylene blue, have been found. These Negri bodies (see Plate IV) are minute ($1–23\mu$ in diameter) round or oval objects whose exact nature is unknown. Negri and others regard them as minute protozoa, the causative agents of the disease.

Of this there is no other evidence than their constant presence in the nervous system in rabies. The diagnosis of rabies in the dog is now confirmed by the examination of the nervous system and the demonstration of these bodies. For this purpose a bit of the central nervous system, preferably the cerebellum or the hippocampus major, is crushed and a thin smear prepared. This is hardened by immersion in wood alcohol for 2 or 3 minutes and then stained. Weak solutions of eosin and methylene blue (or Jenner's stain) may be used as for blood. The Negri bodies appear as pink or reddish inclusions in the nerve-cells.

Symptoms.—The period of incubation in man is usually from six to eight weeks. It may, however, be as short as two weeks or as long as three months. The wound heals promptly and the scar presents no unusual appearances. Infection may occur without a wound.

The symptoms may be divided into three stages:

(1) A PREMONITORY STAGE, lasting a day or two, during which there are restlessness, anxiety, depression and insomnia and often also some pain or soreness in the cicatrix.

(2) THE SPASMODIC STAGE, in which there are intense excitement and restlessness, together with frequent severe and extremely painful spasms of the muscles of the larynx and pharynx. These are excited by any attempt at swallowing or by the mere sight or thought of water or by any slight external stimulus such as a sound or touch. The spasms often involve the muscles of respiration and may amount to a general convulsion. The mind is usually clear, but there may be maniacal outbreaks. The temperature is somewhat elevated and the pulse grows progressively rapid and weak. This stage lasts one or two days and is succeeded by—

(3) A TERMINAL STAGE OF PARALYSIS, which, in man, lasts but a few hours, and in which the spasms cease, prostration becomes extreme, unconsciousness develops and death ensues. The disease is almost invariably fatal within four or five days.

Diagnosis.—Hydrophobia may be closely simulated by a curious hysterical condition (lyssophobia) which occasionally develops in those who have been bitten by a dog and who are in terror of rabies. There may be inability to swallow water, spasms and various other hysterical manifestations. Fever is lacking. The symptoms subside under suitable moral treatment.

Treatment.—PROPHYLAXIS.—Rabies can be stamped out by the proper muzzling of all dogs and the destruction of such as are ownerless.

The wound should be immediately cauterized by strong carbolic acid or should be excised.

It is of the utmost importance to know whether the offending dog is really suffering from rabies. He should, therefore, be confined, rather than shot, since if rabic he will surely die within a very few days. If shot the medulla should be removed and a small portion of it emulsified in glycerine or broth and injected beneath the dura mater of a rabbit. If rabies is present the animal will die of paralysis (dumb rabies) in

about two weeks. The demonstration of the Negri bodies as above described is now regarded as entirely reliable.

PREVENTIVE INOCULATION.—Pasteur's method consists in the daily subcutaneous injection of emulsions of the spinal cords of inoculated rabbits, the virus having been attenuated by desiccating the cords for a certain number of days. This method is almost always successful if the incubation period be sufficiently long to allow the proper number (12–25) of injections to be made.

After the disease has developed, treatment is merely palliative and consists in attempting to control the spasms by the use of hyoscine hydrobromate (gr. 1/100), morphine or chloroform.

ANTHRAX

(Splenic Fever; Malignant Pustule; Wool-Sorters' Disease)

Definition.—Anthrax is a severe infectious disease of certain domestic animals, which is due to a specific bacillus and which is occasionally communicated to man.

Etiology.—The disease occurs chiefly among cattle, sheep, horses and other herbivorous animals, and in certain countries and regions exists as a wide-spread and destructive plague. It is not common among the animals of this country.

The specific germ is a very large, non-motile rod—the BACILLUS ANTHRACIS—which under certain favorable conditions forms extraordinarily resistant spores, and these spores seem to be the chief agents in the transmission of the disease. The bacilli are found in the local lesions, in the blood and tissues, and in the discharges. In man, anthrax is seen almost exclusively among those who are brought in close contact with such animals, or with hides, wool, etc., *e.g.*, butchers, drovers, tanners, wool-sorters, etc. In New York a number of cases have been seen among 'longshoremen. Inoculation occurs either through abrasions of the skin (external anthrax), or through inhalation or ingestion of the virus (internal anthrax).

Symptoms.—The disease appears in two chief forms:

(1) EXTERNAL ANTHRAX OR MALIGNANT PUSTULE.—Inoculation occurs usually upon the exposed parts, such as the face or hands. At the point of infection there appears a small red papule, which rapidly increases in size and develops an indurated, inflamed base. At the summit of the papule a vesicle forms which soon becomes a blackish, necrotic eschar and around this is often formed a circle of secondary vesicles. These local appearances are quite characteristic. In the course of two or three days the papule develops into a large carbuncle-like swelling around which there is much induration and inflammatory edema. The neighboring lymph-glands are swollen and tender. During this period there is usually a moderate rise of temperature with some constitutional disturbance. If the disease is allowed to progress the inflammation

spreads rapidly, the bacilli enter the circulation, the spleen becomes much enlarged and the patient usually dies, on the fourth or fifth day, with all the symptoms of an intense septicemia. Occasionally no well defined pustule forms and there is only a rapidly spreading local EDEMA with early septicemia and death.

(2) INTERNAL ANTHRAX.—This is less common than the external form and results either from infection of the gastro-intestinal tract by the ingestion of the flesh of infected animals, or from infection of the respiratory tract by inhalation.

(a) *Intestinal Type.* (*Mycosis Intestinalis.*)—There are violent gastro-intestinal symptoms, as well as those of septicemia, and the cases are usually fatal within a very few days.

(b) *Pulmonary Type.* (*Wool-Sorters' Disease.*)—The symptoms are those of a bronchitis or a bronchopneumonia with very severe septicemic constitutional symptoms.

Diagnosis.—Malignant pustule is usually readily recognized by its characteristic appearances and by the finding of the specific bacilli in the fluid from the vesicles. The diagnosis of internal anthrax is, however, very difficult. The disease may be suspected from the occupation of the patient, but a positive diagnosis can hardly be made except by finding the bacilli in the circulating blood.

Prognosis.—The prognosis of internal anthrax is very bad. In malignant pustule, if the condition is recognized early and treated radically, most of the cases recover.

Treatment.—The malignant pustule should be promptly and freely excised and the wound cauterized thoroughly. The treatment of internal anthrax is that of septicemia.

ACTINOMYCOSIS

Definition.—A chronic disease of the type of the infectious granulomata, common in cattle and occasionally seen in man, which is due to the STREPTOTHRIX ACTINOMYCES or ray fungus.

Etiology.—The disease is frequent in cattle, where it causes a sarcoma-like tumor of the jaw, known as "lump-jaw," and is also found in horses and swine. Infection seems to occur through the food. The ray fungus belongs to a group of organisms closely allied to the bacteria. The germs are found abundantly in the pus from the granulomatous tumors and appear in the form of yellowish bodies ("sulphur grains") of pin-head size, which consist of masses of radiating threads and filaments, many of which show bulbous extremities. In man the organism probably finds entrance through carious teeth.

Morbid Anatomy.—The lesions are not unlike those of tuberculosis. The tumors vary greatly in size and are made up of lymphoid, epithelioid and giant cells. In some instances they tend to the formation of much connective tissue and in others they break down and form chronic abscesses and suppurating sinuses. In half of the cases the lesions occur in

the mouth, throat or neck.   Less frequently they are found in the intestines or lungs and rarely also in the liver, brain, skin and other tissues.

Symptoms.—Most commonly there is a slow growing tumor about the lower jaw or in the neck, which eventually suppurates and forms fistulous openings.   In the lungs the symptoms may be those of fetid bronchitis, of bronchopneumonia or of chronic abscess.

In the intestines there may be tumor or abscess formation.   Sometimes the picture is that of a chronic pyemia.

The DIAGNOSIS can be made only by finding the characteristic sulphur grains in the pus or in the sputum.   The course of the disease is very chronic and most of the cases eventually succumb.

Treatment.—Whenever possible, this should be surgical.   In the internal forms large doses of potassium iodide should be tried.

DISEASES CAUSED BY VEGETABLE PARASITES OTHER THAN BACTERIA

Apart from the great variety and number of diseases produced by bacteria the investigations of recent years have shown that the human organism may be invaded by other vegetable parasites and diseases of various types produced thereby.

Infections of this kind, while of great scientific interest, at present are so rare as to be of little practical importance.

The lesions produced by these infections are: (1) CUTANEOUS-GRANULOMATA (nodules or tumors composed of granulative tissue), abscesses or ulcerations.

(2) INTERNAL, SUBACUTE OR CHRONIC INFLAMMATIONS producing nodules resembling tubercles, and, like tubercles, breaking down and producing abscesses and chronic suppuration.

The symptoms, whether of the cutaneous or internal forms of the infections, are those of chronic inflammation and suppuration, the lesions closely resembling tubercles, and the resulting infection being consequently commonly mistaken for tuberculosis.

Among the organisms to be classified under this heading may be mentioned:

(1) *Nocardia*, organisms midway between bacteria and moulds.   They resemble bacteria in form, structure and staining properties, while they have the fine branching forms and spores or conidia characteristic of moulds.   They are widely distributed in the air and on grains or grasses.

(2) *Actinomyces or ray fungus.*   (See Actinomycosis.)   Granules apparently indistinguishable from the actinomyces have been found in the tropical affection mycetonia, a chronic inflammation of the foot (Madura Foot) with abscesses and serous formation closely resembling tuberculosis.   The disease is frequent in India and tropical Africa.   A very few cases have been observed in the United States.

(3) *Oidia.* Spherical vegetable organisms closely resembling the yeast fungus or saccharomyces. These have been found in certain rare skin infections, variously denominated blastomycosis or blastomycetic dermatitis, psoro-spermiasis, dermatitis coccidiodes, etc.

(4) *Blastomycetes,* or yeast fungi, found in certain dermatitis.

(5) *Aspergilli or moulds.*

Doubtless, from time to time additions will be made to this list, as we now know that man is subject to many other infections than the common bacterial types. Practically this possibility is to be borne in mind and search made for such parasites in cases resembling external or internal tuberculosis, but failing to show the tubercle bacillus.

IX

DISEASES CAUSED BY ANIMAL PARASITES

The importance of pathogenic protozoa in human pathology has been recognized only since the discovery of the malarial parasite by Laveran in 1880. Year by year the number of pathogenic protozoa is added to. The differentiation of the thousands of varieties of these organisms, a few pathogenic, the greater number never found in man, is attended with much difficulty. In some cases it is uncertain whether the organism belongs to the animal or vegetable kingdom. Any classification at present must be tentative. To illustrate the types of protozoa and suggest the relationship of known pathogenic types, the following classification, abridged from Calkins, is given:

CLASSIFICATION OF PATHOGENIC PROTOZOA

Protozoa are unicellular animal organisms which reproduce by division or spore formation; solitary or united in colonies; free-living or parasitic.

Phylum I.—Sarcodina.—Protozoa with changeable protoplasmic processes or pseudopodia. Amebæ are included in this class.

Phylum II.—Mastigophora.—Protozoa with flagella. Trypanosomes, circomonas, herpetomonas and the like.

Phylum III.—Infusoria.—Protozoa with cilia. Balantidium coli is the only important example of this class.

Phylum IV.—Sporozoa.—Protozoa without motile organs and reproduced by spores, always parasitic. Plasmodium malariæ belongs in this class. (See Fig. 82, *A, B, C, D.*)

MALARIAL FEVER

Definition.—Malarial fever or malaria comprises the group of morbid conditions produced by the presence in the blood of certain protozoal parasites, whose definitive host is the mosquito and whose intermediate host is man.

Etiology.—Malarial fever has been proved beyond doubt to be caused by infection with a specific parasite, the plasmodium malariæ, transmitted from one individual to another by the bite of a genus of mosquito, the anopheles. The spread of malaria in any region therefore demands the presence of some individuals having these parasites in their blood and of the particular mosquito capable of acting as host and transmitting them to man. Where either one of these conditions is lacking malaria must die out; where both are present, malaria, unless rigid precautions are taken, thrives. The conditions long famed as causing malaria are found to be those favoring the life and activity of the malaria-carrying mosquito, the anopheles. The life-history of

the anopheles is the key to nearly all the mysteries which so long sur-
rounded this subject.

MOSQUITOES OF THE GENUS ANOPHELES.—The distinctive features of
these mosquitoes are that both sexes have palpi almost as long as the
proboscis, that their wings are commonly spotted, the body colors brown
and yellow, that at rest body, head and beak are carried in one plane,
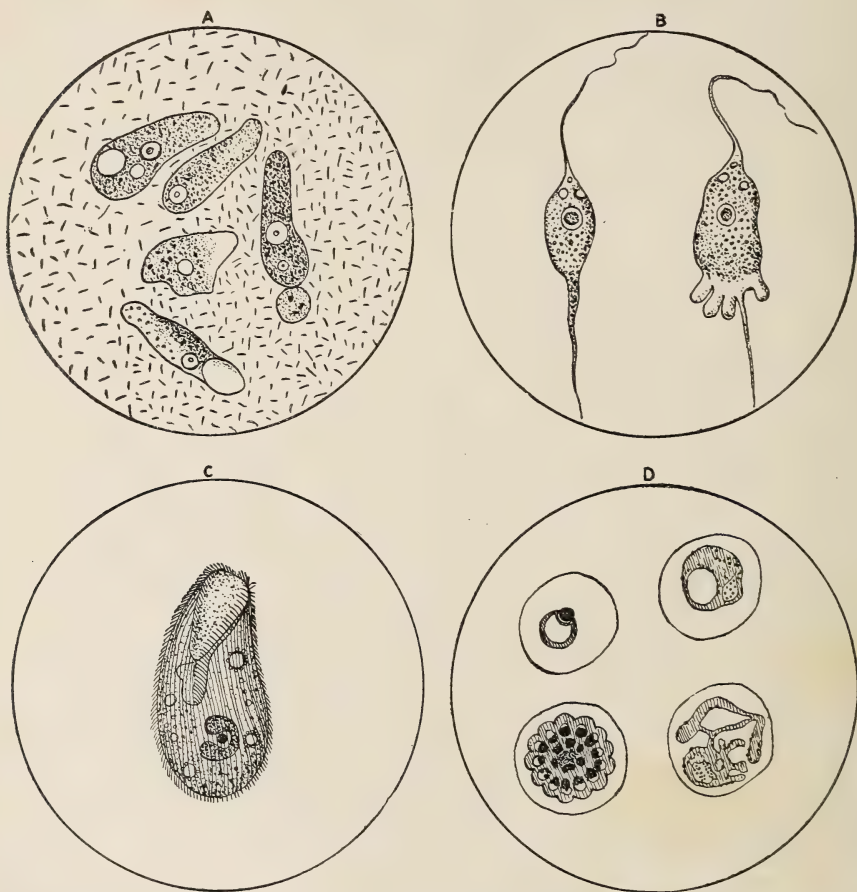
FIG. 82.—Types of protozoa. *A*, Ameba Coli. (After Calkins.) *B*, Cercomonas Crassicanda.
(After De Page and Herouad.) *C*, Balantidium, Entozoön Infusorium. (After Calkins.) *D*, Spor-
ozoa, Plasmodium Malariæ. (After Ewing.)

whereas in other varieties there is a marked angle between head and
body, and finally when resting on any surface the body is not parallel
but at an angle of about 80° to the surface.

The eggs are laid in shallow, stagnant water, each female depositing
from 40 to 100 eggs. In from 3 to 4 days in warm weather the
larval form develops and lies floating on the surface of the water. In
from 16 to 20 days the larva develops into a pupal stage lasting from 3

to 10 days, when the full-grown mosquito emerges. For the development of this mosquito stagnant water and a certain degree of warmth (an average temperature of at least 60°) are necessary. These mosquitoes breed most readily, therefore, in marshes, in the sedgy banks of slow-moving streams, in shallow pools or puddles of water, in rain-water collected in old cans and the like. The anopheles never fly high or far from their birth-place, and the life of any generation is limited to about 20 days. They are inactive during the day, except in darkened or shady places, while they bite most freely during the night. In these facts we find the explanation of the deadly peril of night air in malarial districts, of the miasms that rise from swamps, of the freedom from malaria enjoyed by mountain resorts, of the prevalence of malaria in warm seasons, its subsidence in winter, of the danger of newly-ploughed lands (furnishing puddles), of the advantage of cities and towns especially under modern hygienic conditions. The reduction in the prevalence of malarial fevers brought about by warfare against the mosquito, especially on the Panama Canal zone, is most convincing proof of their malignant activities.

DISTRIBUTION OF MALARIAL FEVERS.—In one or another form malaria is known from the equator to the Arctic Circle, but the tropics have always been the seat of its greatest and most deadly activity. In the temperate zone only the milder forms of intermittent or continuous fever are seen. In England and in Continental Europe excepting Italy malaria is almost unknown.

Season.—In this latitude malaria is seen during the winter only as the recrudescence of an old infection. New cases appear in the spring with the development of conditions favoring the development of the mosquito, and the disease prevails till the coming of winter.

Altitude.—The immunity of mountain regions depends on two factors, the absence of stagnant water and the lower temperature. If conditions are favorable mosquitoes may develop and malaria abound in mountain regions.

Sex and Age.—All ages and both sexes are vulnerable, but malaria abounds among children in the tropics, because the mosquito bites them by preference. The black races enjoy a certain immunity, acquired, according to Koch's observations, as the result of long-continued infection in childhood.

Recurrence.—In persons suffering from latent malarial infections, a chill, fatigue, hunger, or any like influence reducing the vitality may precipitate a new outbreak.

Incubation.—About ten days ordinarily elapse between infection with the parasite and the appearance of fever, but this period may be protracted by various influences.

MALARIAL PARASITE OR PLASMODIUM MALARIÆ

The organism causing malaria is a microscopic body, of varying shape and size, found either in the red blood-cells or the plasma of the

patient. It is classed as a protozoan. It cannot, therefore, be grown upon culture media like bacteria, nor is it found outside the bodies of man or the definitive host, the mosquito. Similar parasites are found in the bloods of various mammals, birds, reptiles, fishes. Hundreds of varieties have already been catalogued. The plasmodium malaria shows a number of different forms, each associated with a different type of fever.

Tertian Parasite (see Plate V).—In its earlier stage this organism is seen as a small, pale ring, with a spot of pigment at some point on the margin, lying within a red blood-corpuscle. In fresh blood it shows active ameboid movement, constantly changing form and putting out pseudopodia. Gradually it increases in size, and numerous pigment granules, dancing actively, appear within it. The blood corpuscles holding the parasite become large and pale, sometimes twice the normal size.

If the blood be deeply stained with Leishmann's or Giemsa's stains the hemoglobin of invaded cells is found to be marked by minute specks, chromophilic particles, "Schuffner's dots." These are distinctive of the tertian infection.

With further enlargement of the parasite the red blood-cell disappears, is destroyed, and the parasite is set free in the plasma. It then appears as a spherical body larger than a red blood-cell, approximating in size the white cells, with several pigment granules near the center. In this stage two different forms related to the further development of the parasite, as intracorporeal or extracorporeal, can be distinguished.

(a) Certain of these large spherical forms develop evidences of fission and finally break up into a number (15–26) of small spherules, each with a minute nucleolus, and each of these little spherules undergoes in turn the evolution above described, the INTRACORPOREAL, ENDOGENOUS OR ASEXUAL CYCLE of the malarial parasite. The entire cycle of the tertian parasite occupies forty-eight hours.

(b) EXTRACORPOREAL CYCLE.—When a number of these spherical bodies are taken into the stomach of a mosquito which has drawn the blood of a malarial patient, they are quickly differentiated into two distinct types, one hyaline, the male element, and the other granular, the female element. The male spherules then put out actively moving flagella (pseudopodia). These flagella finally break away from the parent cell and make their way, apparently by their own activity, into one of the granular female cells and there disappear. This newly impregnated spherule then undergoes a series of changes resulting in the production of a lance-shaped or vermicular body possessed of distinct powers of penetration. This body makes its way into the muscular wall of the stomach of the mosquito, and there undergoes development into a cyst-like form, which subdivides into a number of spindle-shaped bodies contained within a capsule. In the end the capsule ruptures and the spindle bodies are discharged into the body cavity of the mosquito. Thence they make their way, probably by the blood stream, to the salivary glands of the insect. From the salivary glands the proboscis opens by

PLATE V.

The Parasite of Tertian Fever. (Drawn by Mr. Brödel for Thayer and Hewetson's paper, The Malarial Fevers of Baltimore, Johns Hopkins Hospital Reports, Volume V. We copy the original legend.)

    1. Normal red corpuscle.

2, 3, 4. Young hyaline forms. In 4, a corpuscle contains three distinct parasites.

5, 21. Beginning of pigmentation. The parasite was observed to form a true ring by the confluence of two pseudopodia. During observation the body burst from the corpuscle, which became decolorized and disappeared from view. The parasite became, almost immediately, deformed and motionless, as shown in Fig. 21.

6, 7, 8. Partly developed pigmented forms.

    9. Full grown body.

10–14. Segmenting bodies.

    15. Form simulating a segmenting body. The significance of these forms, several of which have been observed, is not clear to the writers, who have never met with similar bodies in stained specimens so as to be able to study the structure of the individual segments.

16, 17. Precocious segmentation.

18, 19, 20. Large swollen and fragmenting extra-cellular bodies.

    22. Flagellate body.

23, 24. Vacuolization.

The Parasite of Quartan Fever.

    25. Normal red corpuscle.

    26. Young hyaline form.

27–34. Gradual development of the intra-corpuscular bodies.

    35. Full grown body. The substance of the red corpuscle is no more visible in the fresh specimen.

36–39. Segmenting bodies.

    40. Large swollen extra-cellular forms.

    41. Flagellate body.

    42. Vacuolization.

PLATE V.

The Parasite of Tertian Fever.
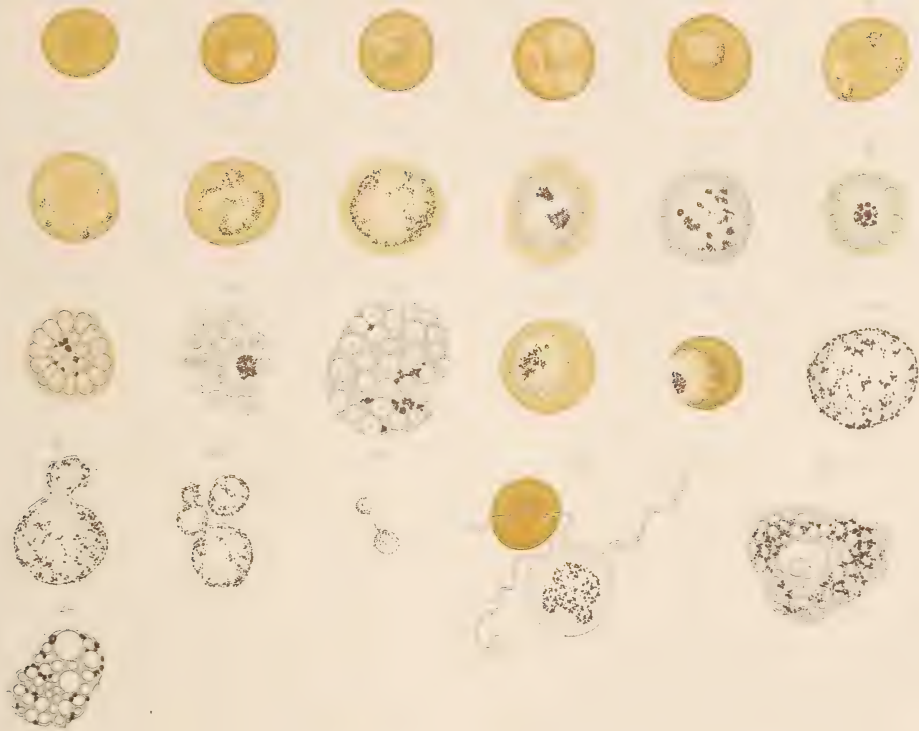
The Parasite of Quartan Fever
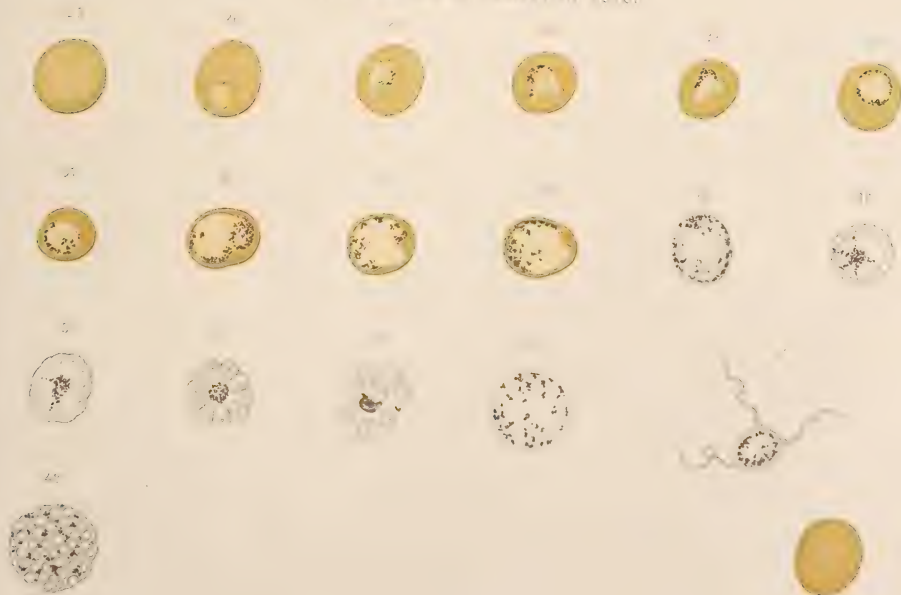
a long duct and through this channel these spindle bodies are discharged into the blood of a person bitten by the infected mosquito. In the blood of the new host the spindle bodies enter the corpuscles and there develop into the intracellular forms already described.

The extracorporeal cycle of the malarial parasite occupies about two weeks' time.

Quartan Parasite.—This organism in its earliest forms is a small round body in a red cell, smaller than the tertian variety. Its ameboid movement is much less active, and as soon as it becomes pigmented, ceases entirely. The pigment granules are coarse and the total amount is large. Segmentation occurs in a manner suggestive of the petals of the daisy around a relatively large mass of centrally placed pigment. It finally divides into eight to ten segments, as compared with the 16–26 of the tertian parasite. The quartan organism does not cause marked enlargement of the cells in which it lies—but when mature completely fills them. The ''daisy'' segmenting form is more frequently seen in the peripheral blood than the corresponding phase of other parasites. By these characters the quartan parasite can ordinarily be readily distinguished. Its complete intracorporeal cycle occupies 72 hours.

Estivo-Autumnal Parasite (see Plate VI).—Parasite of malignant malarial fevers. The earliest form of these is a minute unpigmented ring, discoverable only with difficulty, in the red cells. Multiple infection of individual corpuscles is more common than with other forms. It is also characteristic that the parasites are relatively infrequent in the peripheral circulation, but are found in great numbers in the capillaries and small arteries of the deep viscera (especially the spleen) and the bone marrow. They are, therefore, sometimes discovered only after death. These parasites tend to produce a peculiar shrinking of the infected corpuscles, so that many of these become crenated and very dark, the so-called ''brassy bodies.'' The larger pigmented forms of this parasite, corresponding to the fully developed tertian or quartan varieties, are rarely seen in the peripheral blood. Instead, there appear either in the red cells or free in the plasma certain crescentic forms or crescents, which are characteristic of the organism. These crescents are longer than the red cells, slightly curved, the ends rounded, with definite pigment masses near the center of the body. They may appear as though attached to a red cell, a part only of the body of the latter showing in the bay of the crescent, or may be free in the plasma. Contrary to the behavior of the other forms of the malarial parasite, they do not at once disappear from the peripheral blood on the administration of quinine, but may persist for from three to six weeks. The life span of the estivo-autumnal organism, owing to the difficulty of finding mature forms, is much less accurately known than those of the tertian or quartan forms, but is calculated at from 24 to 48 hours.

Both the quartan and estivo-autumnal forms undergo an extracorporeal cycle analogous to that of the tertian parasite.

Morbid Anatomy.—The milder malarial fevers seen in this latitude

28

—quotidian, tertian or quartan—are rarely fatal and their anatomical changes must be inferred from those of pernicious malarial fever or cachexia. In these conditions the skin and mucous membranes are very pale, and the skin has an icteric tint, but the scleræ are clear. The blood, besides the presence of the parasites, shows a marked secondary anemia, by reduction both of cells and hemoglobin, particularly the latter. The leukocytes are usually moderately increased—10,000 to 15,000—with an increase in the percentage of large mononuclears. The leukocytes frequently show pigmentation with hemozoin derived from the parasites. The plasmodia are often found most abundantly in the blood of the spleen, liver, or bone marrow.

The spleen is greatly enlarged and very soft, diffluent. It is often deeply pigmented.

The liver is large, soft, congested and also pigmented. Two forms of pigment are found in both these organs—(a) hemozoin derived from the parasites; (b) hemosiderin from the hemoglobin of destroyed red cells. Other organs, the lungs, kidneys, intestines, may show like pigmentation.

Acute nephritis, myocardial degeneration with dilatation of the heart, pneumonia, or empyema and dysenteric lesions of the colon may be found.

Symptoms.—INCUBATION.—In experimental infections, the latent period has been from 2 to 10 days. Susceptible persons may, however, dwell in malarial regions for some time before seizure. The United States soldiers, serving in Cuba, as a rule, had been there a month or more before developing symptoms.

For clinical purposes we distinguish four groups of cases: 1. Regular intermittent fevers—quotidian, tertian, quartan. 2. Irregular remittent or continued fevers—estivo-autumnal fever. 3. Pernicious forms. 4. Malarial cachexia.

I. THE REGULAR INTERMITTENT FEVERS

The characteristic feature of these fevers is the recurrence at definite intervals of the malarial paroxysm, marked by chill, fever and sweating. A period of several days or a week of indefinite malaise, with loss of appetite, a coated tongue, and slight headache, precedes the development of typical chills. Once the chills begin they are repeated with almost clock-like regularity at definite intervals, every day (quotidian) or every second (tertian) or third day (quartan). The chills occur most often in the early afternoon, sometimes in the morning, extremely rarely at night. (See Fig. 83.)

In the typical paroxysm three stages are distinguished.

THE CHILL sets in suddenly with a feeling of coldness in the back and extremities. The skin becomes pale and cold, the lips may be blue, the pulse small, feeble and either slow, normal, or quickened in rate, and the patient is greatly prostrated. Soon the teeth begin to chatter and

PLATE VI.

The Parasite of Aestivo Autumnal Fever.
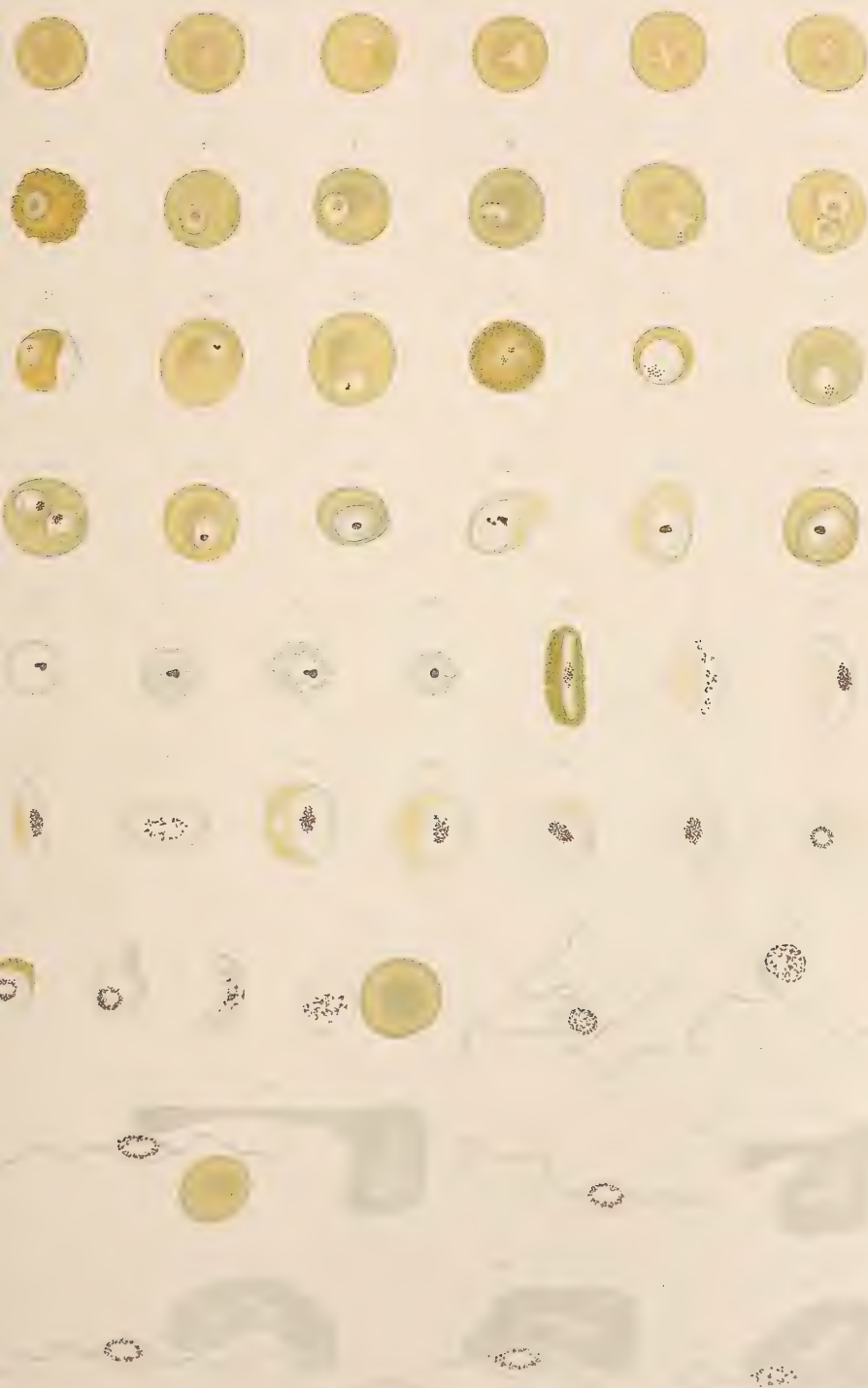
the limbs to shake (rhythmic contractions) ; the pallor, blueness and weakness of the pulse are increased. The surface temperature during this time is low; the internal (rectal) temperature, normal at first, is raised several degrees. Headache may be severe, and nausea and vomiting are frequent. After from one-half to two hours the HOT STAGE succeeds. From the chilly condition the patient quickly passes to one of burning fever; the face flushes, the conjunctivæ become suffused, the skin hot and dry, the pulse rapid, full and bounding. The headache is intense, the patient excited, delirious, stupid or comatose. Nausea and vomiting may occur during this period. The temperature reaches 104°–106° F., or even more. A general erythema or urticaria may appear. This stage lasts from two to six hours and is followed by SWEATING. Perspiration begins on the face and neck and soon breaks out over the whole body and the fever begins to fall. The sweating becomes profuse and drenching. The temperature falls to normal or below, all the signs of dis-
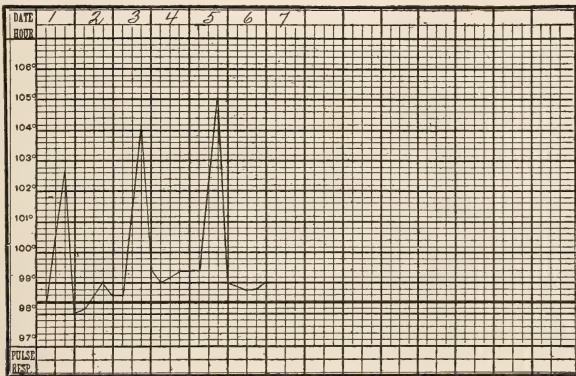
Fig. 83.—Temperature of tertian malaria, three paroxysms.

turbance vanish, and the patient, though weak, feels quite comfortable till the recurrence of the paroxysm. (See Fig. 84.)

More or less definite anemia and enlargement of the spleen regularly develop in patients undergoing these paroxysms. In the first days neither may be notable, but with each recurrence they become more marked and in advanced cases both are extreme. The splenic enlargement of malarial fever is important, the spleen usually being larger than in any other febrile condition, second in size only to the tumors seen in leukemia or Hodgkin's disease. Herpes labialis is often present.

Relation of Paroxysms to Plasmodia.—Each chill is found to be coincident with the maturation of a group of parasites in the patient's blood. The tertian parasite's life-cycle is 48 hours, with a single infection the patient has chills every second day (tertian fever). Similarly the life-cycle of the quartan parasite being 72 hours, the chills would recur every third day (quartan fever). Double infections with the

tertian parasite, one group maturing every twenty-four hours, give rise to daily chills (quotidian fever). The same result may theoretically be brought about by infection with three distinct groups of the quartan parasite. Double infections with the quartan organism give chills on two successive days with a single day's interval.

Course.—Once established such paroxysms may go on indefinitely unless treated effectively. On the other hand, they may spontaneously cease at any time and the patient enjoy an indefinite period of freedom till the lowering of his vitality from any cause (exposure to cold and wet, fatigue, fright, etc.) causes a new outbreak.

Recurrences are very common. These intermittent fevers are rarely fatal, but in the tropics any form of malaria may develop the characters of the pernicious fever.

Diagnosis.—The periodicity. Malarial chills tend to return at the same hour of the day. The enlarged spleen, the rapidly developed
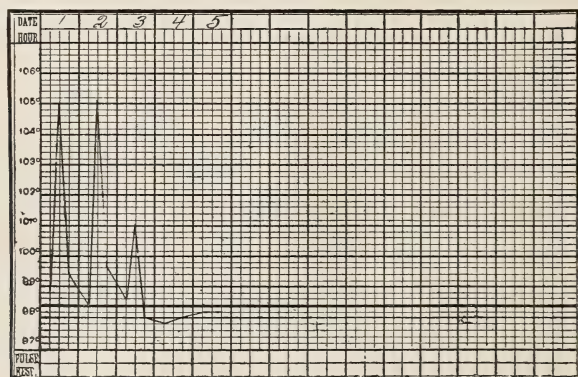
Fig. 84.—Quotidian malaria, daily paroxysms.

anemia make the intermittent fevers easily recognizable. The diagnosis is made certain by the presence of the parasites. These are best sought for about the time of the chill, when they are most mature, but may be found at any time. More than one examination may be required. Quinine should not be given before the examination, for the drug quickly causes the disappearance of the parasites from the peripheral circulation even when it does not check nor stop the paroxysms of fever.

The chills of sepsis such as occur in pyemia or advanced tuberculosis may simulate malaria, but they are never so regular. In pyemia the chills, moreover, recur at irregular intervals day or night. The parasites of malaria are not to be found in the blood. Neither the anemia nor the splenic enlargement, though present, is as pronounced as in malaria; a leukocytosis and increased polynuclear percentage may be shown by the blood, or blood cultures may yield bacteria. There will develop localized symptoms of suppuration in some part or parts.

In advanced tuberculosis chills and sweating may recur each afternoon, but there are local (pulmonary, osteal or glandular) signs of the disease, bacilli in the sputum, and no parasites in the blood.

Finally none of these fevers yields to quinine; the malarial intermittent fevers are promptly cured by it.

II. IRREGULAR REMITTENT OR ESTIVO-AUTUMNAL FEVER

It is extremely difficult to describe the various types of fever which may be caused by infection with the estivo-autumnal parasite. Temperature curves of endless variety may be shown. The fever may be very slight or very high, continued, or remittent, or may come and go in such manner as to make it impossible to classify the curve.

Whatever the type of fever, chilly sensations, or distinct chills may accompany the rises and sweating occur with the falls. The patient regularly becomes markedly anemic and the spleen is notably enlarged. Irregular fever, anemia and a large spleen may be termed the fundamental features.

The estivo-autumnal infections are likely to be persistent and severe. In the temperate regions they constitute the only severe malarial cases. In the tropics, although any malarial fever may become severe and dangerous, the estivo-autumnal or ''crescent'' infections constitute the great bulk of cases of so-called pernicious malarial fever.

Among the more common types of estivo-autumnal fever the following may be cited:

(1) **The Continued Fever Resembling Typhoid.**—The patients give a history of malaise and fever with gastric and intestinal disturbance lasting for one or two weeks. They have a continued fever, possibly more irregular than the usual typhoid chart, but not easily distinguished from it; they suffer from headache, prostration, diarrhea or constipation, pains in the back and limbs; the appearance is that of typhoid fever, and the disease is frequently mistaken for it. The spleen, however, is larger than is ordinarily met with in typhoid, the anemia is more marked, there are no rose spots, no Widal reaction, no bacilli in the blood, but on examination (repeated, if necessary) the parasites, especially the crescentic forms, are found.

(2) **A Remittent Type.**—This differs from the latter only in the fact that the morning recessions of the fever are more marked. From the fact that this type of fever is specially often accompanied by severe vomiting and purging and possibly jaundice, the term bilious remittent is often used.

(3) **Irregular Intermittent Types.**—Various intermittent fevers, resembling but not conforming to the type of ordinary quotidian, tertian or quartan fever, may be produced by crescent infections.

In these irregular types chills, fever and sweating may occur, but these symptoms are much less marked and less characteristic than in the simple tertian or quartan infections. The disease is severer and is more likely to attain the so-called pernicious, i.e., a very severe, form.

The notable symptoms in these pernicious types, in addition to the fever, anemia and splenic tumor, are:—

(1) CEREBRAL.—Headache, delirium, hemiplegia or coma may develop. In some cases these intense cerebral disturbances come at the outset, more often during the course of the disease. They are explained by blocking of the brain capillaries by large numbers of the parasites.

(2) GASTRO-INTESTINAL.—Nausea, persistent vomiting, often becoming bile-stained (bilious), and diarrhea, even dysenteric in type, may occur. Jaundice may accompany the severe gastric symptoms. Severe malarial fever accompanied by persistent vomiting and jaundice, therefore, often suggests yellow fever. Similarly many of the patients returning from the Philippines had dysenteric symptoms.

(3) CONSTITUTIONAL COLLAPSE.—At any time during severe malaria symptoms of collapse may develop. In this condition the patient is profoundly exhausted, the pulse rapid, feeble and intermittent, the surface pale, cold and bathed in perspiration, the eyes sunken, the lips and finger tips cyanotic—the algid or, if vomiting and purging be present, the choleraic type of the disease.

III.   MALARIAL CACHEXIA

As the result of repeated attacks of malarial fever, especially of the estivo-autumnal type, a condition of extreme anemia and profound exhaustion, with enormous enlargement of the spleen (ague cake), may be produced. The patients present the pallor, palpitation, dyspnea, constipation, or diarrhea, possibly edema of the ankles, characteristic of profound anemia. The spleen projects below the edge of the ribs and may occupy the whole left side of the abdomen. The urine may show albumin and casts.

The explanation of the irregularity of the symptoms in estivo-autumnal fever appears to be that there are a number of different groups of organisms, each maturing at a different time. The toxins produced by these organisms seem also to be of a peculiarly virulent type.

Sequelæ.—Profound mental disturbances, explained by the blocking of the capillaries of certain cortical regions by parasites, may occur. Melancholia, mania, or delusional insanity may be met with. Chronic nephritis, severe anemia and chronic enlargement of the spleen are also possible results of severe or repeated attacks of fever.

Diagnosis.—In New York the diagnosis of malarial fever is extremely easy. The great majority of cases are typical tertian or quotidian fever; quartan fevers are very rare. The estivo-autumnal infections are of the typhoid type and without laboratory aids may easily be mistaken. But if the Widal reaction is tested and blood cultures are employed, the blood examined for plasmodia, and the effects of quinine tried, there is little chance of error.

Mild irregular fevers due to malaria may easily be overlooked if blood examinations are not made.

In the tropics, on the other hand, malaria is protean in type and

easily confused with typhoid or yellow fever, cholera, dysentery, sepsis or tuberculosis. It is, therefore, clear that mistakes can be avoided only by having the facts constantly in mind and employing the laboratory methods which are essential to diagnosis. Long tables of differential diagnosis are much less important than the examination of the blood for parasites, or for typhoid bacilli, or the stools for ameba or the cholera vibrio, etc. The administration of quinine should not be begun till the blood has been examined for the parasites.

The suggestive features of all malarial fevers are the fever, anemia, and enlarged spleen.

Prognosis.—The malarial fever of these latitudes is benign. It yields promptly to quinine in moderate doses. In neglected cases the anemia and prostration may become profound.

In the South and in the tropics malaria is a severe and often deadly disease, yet the report of Colonel Gorgas for the year 1909 shows that of 10,000 malarial patients on the Isthmus of Panama but 52 died during that year—as remarkable a tribute to the advantages of early diagnosis and adequate treatment as is the limitation of the disease to the sanitary methods employed.

Prophylaxis.—The prevention of malaria follows three general lines: (1) the destruction of the breeding places of the insects and the insects themselves.

The measures required in the destruction of the breeding places are the drainage of marshes, pools, puddles, etc., or the covering with petroleum of such stagnant waters as cannot be drained, the clearing away of brush and under-growth about habitations, the removal or screening of all waters in cisterns and the like.

(2) The prevention of infection of the mosquitoes by preventing their approach to malarial patients by screening hospitals or keeping netting over the beds. (3) Protection of individuals in malarial neighborhoods by screening the houses, and by the periodic administration of quinine, two or three grains thrice daily.

Treatment.—For many mild cases of intermittent fever, an initial dose of calomel and 5 grains of quinine sulphate in capsule three times a day is all that is required. For severer cases rest in bed and a light or fluid diet may also be ordered. Some prefer to give the quinine in one large dose, 15–20 grains, 3 or 4 hours before the time for a chill. Small doses of quinine should be given for many weeks after the cessation of the chills and repeated each spring and summer to avoid return.

In the estivo-autumnal fevers, especially in the pernicious forms, an initial purge is followed by one large dose of quinine, and then 5 grains every 3 or 4 hours. As much as 40 grains a day may be required for several weeks before the fever is conquered. If vomiting occurs or the patient cannot swallow, the quinine may be given hypodermatically, the bihydrochloride or bihydrobromide being used because of their ready solubility. After recovery the patient should from time to time take quinine in small doses, 2 grains thrice daily, for a week at a time to

prevent relapses. Anemia must be treated by iron and arsenic. Malarial cachexia calls for removal to a healthful climate, baths and saline mineral waters such as are had at various watering places, and the proper treatment of the anemia.

BLACK-WATER FEVER—HEMOGLOBINURIC FEVER

Definition.—A fever of tropical and subtropical countries whose chief symptom is the passing of hemoglobin in the urine.

Etiology.—Two radically divergent views are held. (1) That black-water fever is merely a pernicious malarial fever, the outbreaks of which, for reasons unknown, are closely associated with the taking of quinine. (2) That the fever is not malarial but is caused by infection with some as yet unknown organism.

The fever is met with in all tropical and sub-tropical regions, its distribution being that of severe malarial fevers. It has occasionally been observed in the Southern States. Tropical Africa and India are the favorite seats of the disease. It is never found except in people who have had malaria, especially in those who have had repeated attacks. In the tropics it frequently occurs in epidemic form.

Morbid Anatomy.—The liver and spleen are enlarged, soft and show degeneration with the presence of hemozoin and hemosiderin. The kidneys are enlarged, congested, the tubules perhaps blocked by hemoglobin infarcts, and pigment present in the capillaries and cells.

Symptoms.—The period of incubation is entirely unknown. The onset is sudden with chill, fever and sweating, as in malaria. With the paroxysm there is aching in the loins or over the bladder and the urine passed is found to be very dark, a deep red or black. Jaundice quickly develops. Persistent vomiting of bile-stained fluid is common.

The course of the disease is extremely irregular. A single paroxysm may be all, or the fever may continue like an atypical malaria for days or weeks, the hemoglobinuria, jaundice and vomiting following the rise and fall of the fever. During the attacks both spleen and liver are enlarged.

The urine in black-water fever is deep red or black, with a heavy brownish-gray sediment. Albumin is present in large amount, and hyaline and granular casts and much granular material are found. Red blood-cells are few or absent. Spectroscopic examination of the urine shows the double band of oxyhemoglobin or the bands of methemoglobin.

Diagnosis.—The disease must be distinguished from paroxysmal hemoglobinuria, yellow fever, malarial fevers and icterus gravis. Paroxysmal hemoglobinuria bears no relation to previous malarial attacks and is a milder affection. So far as the initial attack is concerned the phenomena of hemoglobinuric fever and paroxysmal hemoglobinuria are the same.

The only distinguishing point from certain irregular types of malarial fever is the absence of the plasmodia. Some authors insist that the

parasites can be found by sufficiently careful examination and that the disease is malarial.

From yellow fever the disease is distinguished by the severity of onset, the early jaundice, and the hemoglobinuria. Hematuria occurs in yellow fever, but blood pigment is not found without the red cells.

Prognosis.—The mortality is high, 25 per cent. Many attacks are, however, mild, and may be often repeated without fatality. The severity of the disease varies in localities and in different epidemics.

Treatment.—This must vary with the conception of the disease. Some treat it as severe malaria by an initial dose of calomel followed by heroic doses of quinine, as much as 120 grains per day. Others say that quinine causes the hemoglobinuria and withhold it.

Rest in bed, the administration of fluids, especially water, according to the condition of the stomach, enemata of physiological salt solution or hypodermoclysis and cups or hot fomentations to the loins constitute the rational treatment of the disease. A combination of 10 grains of sodium bicarbonate and bichloride of mercury gr. 1/30, given every two hours, is said to be of value.

SYPHILIS

(Lues)

Definition.—Syphilis is a specific infectious disease of very chronic nature which is communicated either by direct inoculation or by hereditary transmission. The acquired form is marked (1) by a local initial lesion; followed after a few weeks (2) by constitutional symptoms and eruptions upon the skin and mucous membranes (secondary lesions), and, after a much longer interval, usually by (3) specific growths in the various viscera and tissues (tertiary lesions). In the hereditary form the initial lesion is lacking.

Etiology.—Syphilis has been known for many centuries. It is worldwide in distribution, attacks all races and may be acquired at any age. In this country it is especially prevalent among the negroes. The specific organism of syphilis is, in all probability, the protozoan parasite first observed by Shaudinn and Hoffman in 1905. The organism, a delicate spirochete (spirocheta pallida), is found generally in the local lesions and in the blood of the patient. While the organism is regularly found in syphilis, and not in other conditions, in man, and also in animals (monkey, rabbit) inoculated with syphilis, it has not yet been isolated by cultural methods and, therefore, the final proof of its causative relation to the disease is still lacking.

Modes of Infection.—The disease is communicable usually during its primary and secondary stages. The virus resides in the secretions of the open sores, in the blood, saliva, etc., and gains entrance usually through abrasions in the skin or through a mucous membrane.

Inoculation.—This occurs in most cases upon the genitalia during sexual intercourse. It may occur, however, upon the lips, tongue, tonsils,

nipples, fingers, etc., by direct contact, as in kissing, suckling, vaginal examination, etc., or by means of infected implements such as razors, dental instruments, pipes and eating utensils. Inoculation has occurred rarely in the operations of vaccination, tattooing and circumcision.

Hereditary Transmission.—The chances of transmission are greatest when both parents are diseased. A syphilitic man or woman may, however, transmit the disease to the offspring without the other parent being infected. A healthy woman who has borne a syphilitic child is herself immune from infection from the child, although she presents no evidence whatever of the disease (Colles' Law). If a woman become infected after conception the child may or not develop the disease (placental transmission).

One attack of syphilis usually provides immunity for life. Only rarely have instances of a second infection been recorded.

Morbid Anatomy.—The initial lesion (primary sore or chancre) forms at the point of inoculation. There are infiltration of the skin with round cells, proliferation of connective-tissue cells and often the formation of giant cells. The neighboring small arteries are the seat of an obliterating endarteritis. The adjacent lymph-glands show hyperplasia and induration. The SECONDARY LESIONS include various skin eruptions, mucous patches, warts and condylomata, iritis, etc.

TERTIARY LESIONS.—The most characteristic of these is a circumscribed granulomatous growth, called GUMMA or syphiloma, varying in size from a millet seed to an egg, and found in the skin, mucous membranes, bones, muscles, and the various viscera. These growths, which resemble closely the specific growths of leprosy and tuberculosis, usually undergo caseation at the center and sclerosis at the periphery, but in the skin and mucous membranes they frequently soften and ulcerate. Diffuse fibrosis of the viscera, amyloid degeneration and arteritis also occur as late lesions.

ACQUIRED SYPHILIS

Symptoms.—The INCUBATION PERIOD is usually from three to five weeks.

PRIMARY STAGE.—The initial lesion or chancre appears as a small red or purplish papule which slowly enlarges, becomes dense and indurated at its base and margins, and usually undergoes superficial ulceration. It is almost always single. The adjacent lymph-glands also show induration and enlargement.

SECONDARY STAGE.—This is marked chiefly by constitutional symptoms and by eruptions upon the skin and mucous membranes. The symptoms develop between six and twelve weeks after the appearance of the chancre. There are FEVER, usually slight and transient, but sometimes severe and of a continued, remittent or intermittent type; HEADACHE, usually worse at night; PROSTRATION; ANEMIA; and NOCTURNAL PAINS in the bones and joints.

Skin Eruptions (Cutaneous Syphilides).—These are of various

types, all of which tend to be symmetrical in distribution and free from itching and pain. The earliest form is the syphilitic ROSEOLA—macular spots of pink or reddish color seen chiefly on the abdomen and chest. Later rashes may be papular, pustular, squamous, etc. Of these the papular syphilide is the commonest. It has a coppery or ''raw-ham'' color, is apt to invade the forehead (corona veneris), scalp, face, the region of the genitals and the palms of the hands and soles of the feet, and usually lasts for many weeks. The pustular eruption may closely resemble that of small-pox; the squamous, that of psoriasis. In the moist skin about the anus, perineum and genitals the papules form large, flat growths with excoriated surfaces (flat condylomata).

THE MUCOUS MEMBRANES.—Sore throat is common. It may be due to simple hyperemia, to ulcers upon the tonsils or pharynx, or to MUCOUS PATCHES. These are slightly excavated areas, covered by a grayish pellicle, and seen on the tonsils, fauces, tongue, inner surface of the cheeks, and about the vulvar and anal orifices. Hypertrophy of the papilla may produce the so-called syphilitic wart or papillomata. The larynx may be inflamed and ulcerated.

Other Symptoms.—General, slight enlargement of the lymph-nodes is usual, as is also alopecia. Iritis, retinitis, periostitis, and onychia are occasional symptoms.

The symptoms of the secondary stage usually last from six months to a year, although with energetic treatment they may sometimes subside within a few weeks. They vary much in number and severity. Certain cases are marked by a very unusually severe and intractable course (malignant syphilis). The disease is infectious throughout this period.

TERTIARY STAGE.—Tertiary symptoms are altogether wanting in the great majority of the patients. In untreated cases they develop in from 20 to 25 per cent. In those thoroughly treated the percentage is reduced to five or less. An interval of two or three years usually elapses between the secondary and the tertiary manifestations, but the latter may not appear for ten or even twenty years.

THE SKIN ERUPTIONS are scanty, asymmetrical and tend to involve the deeper layers of the skin and to ulcerate. They may be tubercular, serpiginous, bulbous or rupial in type.

In the EYE iritis, retinitis or chorioiditis may occur.

Nodular thickenings of the periosteum of the long bones, sternum and skull are common.

GUMMATA occur in the subcutaneous and submucous tissue, and in the muscles, bones and internal organs. In and beneath the skin and mucous membranes they are prone to soften and ulcerate. In the deeper tissues they usually undergo sclerosis (see Fig. 85).

Syphilis of the Digestive System.—Gummata occur not infrequently in the soft palate and pharynx. Syphilis of the esophagus, stomach and intestines is very rare. In the RECTUM gummata sometimes occur (almost always in women) and eventually lead to cicatricial stricture. The LIVER is often attacked both in acquired and in inherited syphilis. The lesions

are of three types (a) a chronic perihepatitis, (b) diffuse interstitial hepatitis (syphilitic cirrhosis) and (c) gummata. These latter vary much in number and size and are usually associated with nodular enlargement of the organ. These caseous masses sometimes undergo softening and suppuration but usually they become sclerotic and often leave deep and deforming scars. The symptoms of liver syphilis are usually those of cirrhosis, although the gummatous nodules may closely simulate a malignant growth. Often there are no symptoms whatever.

In addition to the above lesions syphilis not infrequently results in AMYLOID DEGENERATION of the liver, spleen, kidneys, etc.

The Vascular System.—The HEART muscle may be the seat of gummata or of a diffuse fibrosis, or there may be an obliterating endarteritis of the coronary arteries. The symptoms may be those of failing compensation or of angina pectoris.

FIG. 85.—A gumma in the subcutaneous tissue overlying the patella and surrounded by tertiary ulcers and scars.

The arteries, especially those of the brain, often show OBLITERATING ENDARTERITIS. They may also be the seat of small gummatous nodules in the adventitia (gummatous periarteritis). Syphilis is an important factor in the causation of aneurism.

The Respiratory System.—The nose and larynx are favorite sites for the late manifestations of syphilis. In the NOSE ulcerating gummata often cause extensive destruction of bone, and great deformity.

The EPIGLOTTIS and LARYNX may be the seat of diffuse infiltration or of circumscribed gummata, with ulceration, necrosis of the cartilages, deformity and stenosis from cicatricial contracture, etc. The symptoms are those of hoarseness, aphonia, cough, stridor, dysphagia, etc. The pain, however, in contradistinction to that of tuberculous laryngitis, is usually slight or lacking.

In the TRACHEA and large bronchi gummatous nodes, ulcers and cicatricial strictures occur rarely.

Syphilis of the LUNGS is very rare and has little clinical importance. There may be scattered gummata; a diffuse fibrosis beginning about the root of the lung, or the so-called "white pneumonia" found in still-born syphilitic infants.

The Genito-Urinary System.—KIDNEYS.—Acute nephritis sometimes develops in the secondary stage, and gummata are occasionally found.

Syphilitic ORCHITIS is rather common and occurs as an early tertiary symptom. The testicle proper is enlarged, hard, smooth and painless. The epididymis and vas are rarely involved. An associated hydrocele is not uncommon.

The Nervous System.—Of all the various manifestations of tertiary syphilis those affecting the brain and cord are the most important. The lesions follow three chief types:

(a) DISTINCT GUMMATA, which are commoner in the brain than in the cord; are usually superficially placed, and which give the usual general and focal symptoms of a rather rapidly developing tumor.

(b) GUMMATOUS MENINGITIS, involving chiefly either the pia or the dura mater. In the brain this thickening usually occurs at the base and tends to involve the cranial nerves, either by pressure or by extension of the inflammatory process to the nerve-trunks.

(c) SYPHILITIC ARTERITIS.—This often results in occlusion of the lumen, and in softening or sclerosis of those areas in the brain or cord nourished by the affected artery. In the brain the middle cerebral, or some branch of it, is frequently involved. It is clear that in this last type of lesion the results of treatment must be much less satisfactory than in the other two.

Parasyphilitic Affections.—This term has been applied to certain diseases not actually syphilitic, which nevertheless occur much more frequently in syphilitic subjects than in non-syphilitic. Among these are locomotor ataxia, paralytic dementia, and aneurism. They do not respond to anti-syphilitic treatment.

HEREDITARY SYPHILIS

Symptoms.—A large proportion of syphilitic pregnancies result either in abortion or in the child being still-born at term. Living children rarely show signs of syphilis at birth, but when present these consist of a skin eruption (papular, pustular or bullous), enlargement of the liver and spleen and grave malnutrition, and the infant ordinarily dies within a few days.

EARLY SYMPTOMS.—The first evidences of syphilis usually appear between the second and sixth week (rarely after three months) and correspond to the secondary manifestations of the acquired form. The earliest and most constant symptoms are ''snuffles''—a severe and persistent coryza—and hoarseness. About the same time a skin eruption, which is most often papular but which may be erythematous or eczematous, appears over the face, neck, buttocks, extremities, hands and feet. Fissures occur in the lips, at the angles of the mouth and about the anus, and mucous patches are seen on the mucous membranes and at the mucocutaneous margins.

The constitutional symptoms become marked; there are wasting, anemia, cachexia, slight fever, a loose, wrinkled, sallow skin, pinched features, digestive disturbances, hemorrhages, loss of hair and often disturbances of the nails (onychia) and inflammation and possibly sepa-

ration of the ends of the long bones, such as the femur, humerus, etc. (acute epiphysitis). These symptoms may last for a number of months and during this period the disease is highly infectious.

LATE SYMPTOMS.—These correspond to the tertiary symptoms of acquired syphilis and do not always appear. They may develop at any time up to puberty or even later. They sometimes appear in children who have shown none of the early symptoms. The child is undersized, poorly nourished, wizened, and often mentally deficient. Puberty may be much delayed. The PERMANENT TEETH (second dentition) often show very characteristic abnormalities. The upper central incisors show a crescentic

FIG. 86.—Congenital syphilis, with depressed nasal bones and rhagades in the lips. The girl had also a general adenopathy and a syphilitic periostitis of the tibial.

notch in the cutting edge and the teeth are dwarfed, wide apart and somewhat peg-shaped (Hutchinson's teeth). Gummatous lesions of the skin, mucous membranes, bones, periosteum, and viscera may occur as in the acquired form. Interstitial keratitis is the most frequent eye symptom. There may be deafness. Enlargement of the spleen is very common (see Fig. 86).

Diagnosis of Syphilis.—In the primary and secondary stages this is usually not difficult. The possibility of extragenital chancre is to be borne in mind. The characteristic induration of the base and margins of the sore is present no matter where the lesion is situated.

The manifestations of the secondary stage are usually readily recognized by the association of sore throat, a general enlargement of the subcutaneous lymph-nodes, and skin eruptions, with the other symptoms.

The diagnosis of late syphilis, however, when no satisfactory history is to be obtained, may be difficult or impossible. The skin, nose, throat, skull, shins, testicle, penis, etc., should be searched for scars, nodes or other evidences of old or recent syphilis. In women a history of repeated miscarriages is very suggestive. Prompt and decided improvement under anti-syphilitic treatment may usually be looked upon as proof of the disease, although this test is not infallible.

SYPHILIS

The local lesions of syphilis, the chancre, eruptions, mucous patches, adenitis, etc., are so characteristic that they can ordinarily be recognized at once. The later lesions are more uncertain, especially those of cerebral syphilis. Two diagnostic procedures of the greatest value have recently come into general use.

(1) DEMONSTRATION OF THE SPIROCHETA PALLIDA, the specific organism. In all the lesions of syphilis, the organism can be demonstrated by appropriate measures. To obtain the spirocheta from exposed lesions, such as chancres, ulcers, or mucous patches, the lesion should be carefully cleansed, and then curetted, and a drop of the serum exuding after the curettage examined. The specimen to be examined is dried in the air and hardened in absolute alcohol for one hour, or fixed by heat like a blood-smear. Spirocheta pallida is a long, delicate, spirally-curved organism. The length varies from 4 to 14 microns. The fine spirals vary from 6 to 26 in number, and the organism is curved in its length as well as spirally. The stain now used is a modified Giemsa.

Instead of staining the spirochete, the field may be stained by flooding the slide with india ink (a special ink is furnished by Eimer & Amend, of New York) and then washing and drying it. The spirochete will then stand out as a clear-shining white thread on a black background, the so-called dark field illumination.

(2) WASSERMANN'S REACTION.—It has been shown that the blood serum of one animal species possesses the power of dissolving the red corpuscles of another species, and that this power can be intensified in any animal by repeated injections of an alien blood. This hemolysis is found to depend upon two factors, one of which is destroyed by heating to 56° C. for half an hour, while the other survives. The latter is called the amboceptor because it apparently unites or binds the blood corpuscles and the third substance, the complement. If human red corpuscles be mixed with the serum of a rabbit which has been immunized by repeated injections of human blood, the human cells are promptly dissolved, i.e., hemolysis occurs. If, however, the rabbit serum be previously heated to 56° C. hemolysis does not occur, because the complement has been destroyed. But this complement can be resupplied by the addition of fresh serum of any kind, and the process of hemolysis will be completed. This mixture of red blood-cells, amboceptor, and complement is known as a hemolytic system.

In an exactly similar manner a bacteriolytic system can be built up.

An animal can be immunized by repeated injections of a given strain of bacteria. The blood-serum of the treated animal develops the power of binding and destroying the bacteria. This power depends on the two factors, amboceptor and complement, the latter destroyed by heating to 56° C. for one-half hour. The mixture of bacteria, amboceptor and complement in this case constitutes a bacteriolytic system, in which the complement can be destroyed and restored exactly as in the hemolytic system.

Wassermann's test consists in mixing a hemolytic system devoid of complement, and a bacteriolytic system devoid of complement, and then adding just enough fresh serum to supply complement to one chain, but not to both. Under these conditions it is found that the complement will always be taken up first by the bacteriolytic system, and no hemolysis will occur. If, however, the bacteriolytic system is not properly constructed, that is, if serum does not contain the amboceptor fitted to the bacteria employed, the complement is not bound by this system, but is left to unite with the hemolytic amboceptor and hemolysis occurs. Hemolysis, therefore, becomes the test of the presence of the proper amboceptor in the bacteriolytic chain. In Wassermann's original scheme, sheep red cells and rabbit serum immunized to the sheep corpuscles were used for the hemolytic chain, the serum of the patient suspected of syphilis and an extract of syphilitic liver known to contain the spirocheta pallida as the bacteriolytic chain. When these four substances were mixed in a tube and complement in proper proportion added, if the suspected blood-serum really contained amboceptor (antibodies) as it would if the patient had syphilis, the complement would be taken up, there would be none left to complete the hemolytic chain and the corpuscles remained intact. If the patient's serum contained no amboceptor the complement served to complete the hemolytic chain and hemolysis became evident by the breaking up of the red cells and the coloring of the whole mixture red from the dissolved hemoglobin. When hemolysis did not occur, the red cells sank unharmed to the bottom of the tube and the mixture retained its normal clear straw color.

Sheep red cells		Emulsion of syphilitic liver
Hem	Complement	Bacteri
	O	
lysis	Guinea-pig serum	lysis
Rabbit serum immunized to sheep cells heated to 56° C.		Patient's serum suspected of containing syphilitic antibodies and heated to 56° C.
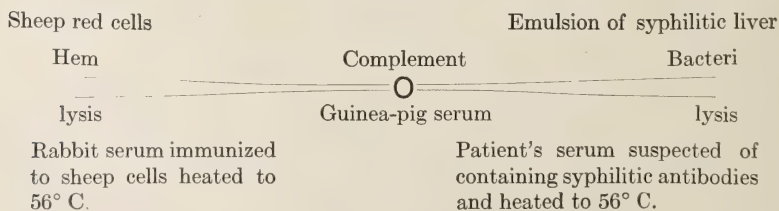
The principle of the test is indicated and the component parts suggested by the used of the words hem-o-lysis and bacteri-o-lysis. There is but one O (complement) available. If one word is completed, the other remains incomplete. If the patient is syphilitic, no hemolysis occurs and vice versa. A failure of hemolysis is, therefore, a positive reaction.

In the Noguchi modification, much used in this country, the arrangement is as follows:

Human red cells	Acetone insoluble residue of an alcohol-ether extract of normal heart muscle or liver.
Complement	
Guinea-pig serum	
Rabbit serum immunized to human blood—heated	Patient's serum to be tested—heated.

Instead of the preparation from the liver of a syphilitic fetus (an organ rich in spirochetæ) various substances have been used, solutions of lecithin, alcoholic extracts of liver, etc.

In the Noguchi modification normal heart muscle or liver is extracted with alcohol, evaporated, the residuum dissolved in ether, evaporated, and the residuum treated with acetone. The part remaining undissolved in acetone is used. It is apparent that the substitution of such substances in the test as well as the finding of the reaction in other conditions, such as leprosy and scleroderma, destroys its specific relation. These irregularities, however, do not seriously affect the value of the test.

The test has been variously modified since its introduction, but the principle remains the same. So many factors are concerned that the utmost accuracy and care are required in carrying it out. Only those who have had months of training and experience with the test are capable of making reliable reports, but in such hands the test has become very valuable. It succeeds in more than 90 per cent. of early cases of syphilis and in varying proportions of others, depending chiefly on the vigor and duration of treatment. The cured patient will not react. In general paresis the percentage of positive reactions has reached 100 in some series.

The failure of a Wassermann test may result from various imperfections in technique. Negative results are, therefore, not proof against the presence of syphilis unless confirmed by repeated trials.

Positive Wassermann tests have been obtained in leprosy and occasionally in some other conditions, scleroderma, but the exceptions are not sufficient to affect the value of the test.

Prognosis.—Except in the case of the congenital form, syphilis is rarely fatal in the early stages. The likelihood of severe tertiary manifestations is greatly diminished by thorough treatment, but cannot be altogether removed. Of these, those affecting the central nervous system are among the most frequent and important.

Prophylaxis.—The problem of the prevention of syphilis is bound up with that of the efficient supervision and regulation of prostitution. Victims of the disease should be thoroughly informed as to its highly infectious nature and should be warned of the danger of transmitting it by sexual contact, kissing, etc., and by means of eating utensils, pipes, etc. Marriage should be forbidden for at least two years after all

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secondary symptoms have disappeared and until a prolonged course of treatment has been undergone.

Treatment.—If any doubt as to the diagnosis exist treatment should be withheld until secondary symptoms appear. Mercury and potassium iodide are specifics; the former being specially useful in the secondary, the latter in the tertiary stage. Mercury may be administered by mouth, by inunction, by hypodermic injection or by fumigation. Gray powder (gr. 1), the protiodide (gr. 1/6–1/4), the bichloride (gr. 1/20–1/12) are the preparations chiefly used by mouth. They are given from three to six times a day. Daily inunctions of mercurial ointment (dr. 1) are an efficient but uncleanly method. Hypodermic injections have many advantages and have been growing in popularity. They should be given aseptically and deep into the muscles of the buttocks, to avoid the possibility of abscess. For this purpose the salicylate of mercury, suspended by careful titration in albolene, one grain to 10 minims, is given in doses of one to two grains every four days. Whatever the method of administration, the mouth and teeth should be kept clean and in good order, to lessen the chance of salivation, and the treatment should be interrupted from time to time. In all, the treatment should be extended over a period of some two years. The late manifestations of syphilis are treated most effectively by the combined use of mercury and the iodide of potassium. The latter is given in gradually increasing doses; beginning with five grains three times a day, and rising to 50 or more grains per dose.

Treatment.—Ehrlich's "606," or salvarsan. Injections of this new drug have proven marvelously efficacious in causing the disappearance of the spirochete from the blood and tissues and bringing about the cure of local lesions. From 0.3 to 0.6 gm. is given by deep intramuscular injection or intravenously.

Various methods of preparing the drug for injection are employed. The following one is recommended. In a graduated, glass-stoppered bottle containing some glass beads the desired dose is put. Fifteen c.c. of hot sterile water are added, and the powder dissolved. Two c.c. of 4 per cent. sodium hydrate solution are next added and the mixture shaken. A heavy yellow precipitate is deposited.

More sodium hydrate solution (1/2–1 c.c.) is added till the precipitate is redissolved. The solution obtained is rather syrup-like and not clear. Sterile water is added to 20 c.c. and 10 c.c. are then injected deeply in each buttock. Care must be taken to avoid the sciatic nerve. The injections are very painful and morphine is frequently required after them. Usually only one dose is given, but in severe cases the injection may be repeated after some days. The possibility of local abscess or sloughing from the injection must be recognized.

To avoid the pain of intramuscular injection or for more immediate effect salvarsan may be given intravenously. The dose is prepared as above except that normal salt solution up to 200 c.c. is added. The perfectly-clear solution resulting is then introduced into the median-basilic vein by a needle just as the ordinary saline infusion.

The use of salvarsan is contra-indicated in any patient having advanced disease of the heart, arteries, kidneys, or nervous system. Particular objection has been urged, because of blindness produced by injections of the closely related atoxyl, to the use of salvarsan in cases of optic atrophy or neuritis. No ill results on the eye have thus far been recorded, but caution should be observed in its use under any of the conditions named.

One injection of salvarsan, it was at first claimed, would cure the disease, but some cases have required 3 or 4 injections to produce any result and others are entirely refractory. Several weeks should elapse between the injections.

After the use of salvarsan, systematic treatment with mercury and the iodides is advisable.

SMALL-POX
(Variola)

Definition.—Small-pox is an acute, highly contagious, infectious disease, characterized by an abrupt invasion, marked constitutional disturbance and a skin eruption which passes through the stages of papule, vesicle and pustule.

Etiology.—The disease has been well known for many centuries and, before the area of vaccination, constituted one of the commonest and most dreaded of the great pestilences.

Distribution.—At the present day, although vastly less prevalent, it occurs in both endemic and epidemic forms over almost the whole world. Its prevalence in any district or country usually bears a direct, inverse relation to the care and thoroughness with which vaccination and isolation are carried out.

Small-pox attacks all ages, even to the fetus in utero. It affects both sexes with equal frequency and is more prevalent during the colder months of the year. Negroes seem especially susceptible. Among aboriginal races the introduction of the disease has always worked frightful havoc.

The EXCITING CAUSE is as yet unknown, in spite of a vast amount of effort expended in its investigation. It is certainly no one of the common pyogenic bacteria present in the pustules.

Although bacilli and cocci are regularly found in the cutaneous lesions of small-pox and sometimes also in the internal organs, these have all been shown to belong to the classes of cocci and bacilli commonly found in the skin and none of them has proven capable of producing the disease in animals. In the epithelial cells of the lesions of small-pox (and also vaccinia) are found certain small bodies of varying size and form. When small-pox is inoculated in monkeys the same small bodies are found in the lesions. Similarly they occur in the lesions produced by inoculating small-pox or vaccinia in the cornea of a rabbit, and can be traced through many successive inoculations from animal to animal. These small bodies are by some declared to be protozoan parasites and the cause of the

disease. The varying forms have been arranged in series supposed to represent stages in the development of the parasite to which the name cytorrhyctes variolæ has been given. Others declare these bodies to be cell-inclusions, the products of degeneration and of no etiological significance. The so-called parasites cannot be isolated or cultivated by any bacteriological methods.

Whatever its cause, the disease is one of the most highly contagious known. The contagious particles are given off from the skin in all stages of the eruption, but especially at the time of the drying of the crusts. They are probably present also in the expired air and in the various excretions. These particles are conveyed by the air, by fomites and through the medium of a third person. The virus is capable of remaining active for months and years in carpets, clothing, toys, etc.

Malignant cases may be contracted from the very mildest ones.

IMMUNITY.—Susceptibility to the disease, in the unvaccinated, seems to be almost universal. One attack usually confers immunity for life. A second attack has, however, in rare instances, been observed.

Morbid Anatomy.—The characteristic lesion is that of the eruption upon the skin and the mucous membrane of the mouth and air passages. Each papule consists of a central area of coagulation necrosis in the deep layer of the epidermis, surrounded by a zone of inflammation. By the gradual accumulation of serum and then of leukocytes, the papule becomes first a vesicle and then a pustule. Many of the pustules involve the true skin and these upon healing leave the familiar depressed scars or pocks. In severe cases parenchymatous changes in the heart muscle and in the liver and kidney are common. The hemorrhagic cases show extravasation of blood into the skin, mucous membranes, meninges, viscera, etc.

Symptoms.—The cases may be divided clinically into four types: the discrete, the confluent, the hemorrhagic or malignant, and varioloid.

THE PERIOD OF INCUBATION is commonly from ten to fourteen days. There are usually no prodromal symptoms.

THE DISCRETE FORM.—The differences between the discrete and the confluent form are seen in the stage of eruption. In the stage of invasion the types usually cannot be distinguished.

Stage of Invasion.—The disease begins suddenly, with one or more CHILLS, VOMITING, intense frontal HEADACHE and pains in the back and limbs, and a prompt rise of temperature to 103° or 104° F. All these symptoms are so constant and so severe as to be quite characteristic. The face is anxious and flushed, the pulse full and rapid and the respiration hurried. Prostration is marked. There may be delirium. In children the initial chill is often replaced by a convulsion. On the second or third day, in 10 or 15 per cent. of the cases, appear the so-called initial rashes. These are quite distinct from the true eruption and last but a day or two. They are usually either scarlatiniform or measly in character, but may be purpuric. They are confined to portions of trunk, thighs and upper arms.

The fever on the second day is very high (104° to 106° F.), but begins to fall on the third day and by the time of the appearance of the true rash is usually only slight.

The RASH appears commonly upon the fourth day. It is first seen on the forehead and wrists and rapidly spreads over face, scalp, extremities (including the hands and feet) and trunk. The eruption consists at first of small red spots which almost immediately become raised (PAPULES) and which have a peculiar, indurated, shot-like feel. They are present often to the number of several hundred and are least abundant over the trunk. The papules increase somewhat in size, and on the fifth or sixth day of the disease change to VESICLES, which soon show a dis-

Fig. 87.—The facial eruption of small-pox at the height of the disease.
From the collection of Dr. S. D. Hubbard.

tinct and characteristic depression at the center (umbilication) and a flattened top. All the vesicles at first are filled with clear serum, but in the course of a day or two (about the eighth day) they become PUSTULES (see Figs. 87, 88), which quickly lose their umbilication and assume a rounded form; the spots first appearing being usually the ones first to become pustular. Around each pustule is an areola of hyperemia so that where the rash is abundant, as upon the face, the skin is greatly swollen, and is tense and very painful. In the course of two or three days the pustules break and begin to dry and by the fifteenth day are usually entirely replaced by yellowish crusts or scabs. These, in the course of a few days, separate and are cast off, leaving behind, in most cases, a discolored spot, but no cicatrix. Only those pustules which involve the true skin as well as the epidermis are followed by permanent scars or pocks.

The eruption is not confined to the skin. Simultaneously, there appear similar spots in the mucous membrane of the mouth, palate, pharynx and often as well in that of the larynx, trachea and bronchi. These spots, instead of becoming typical pustules, usually develop into small, superficial ulcers.

With the appearance of the eruption the temperature falls, the severe constitutional symptoms subside and the patient remains comfortable until the developing pustules cause a second rise of temperature. Its
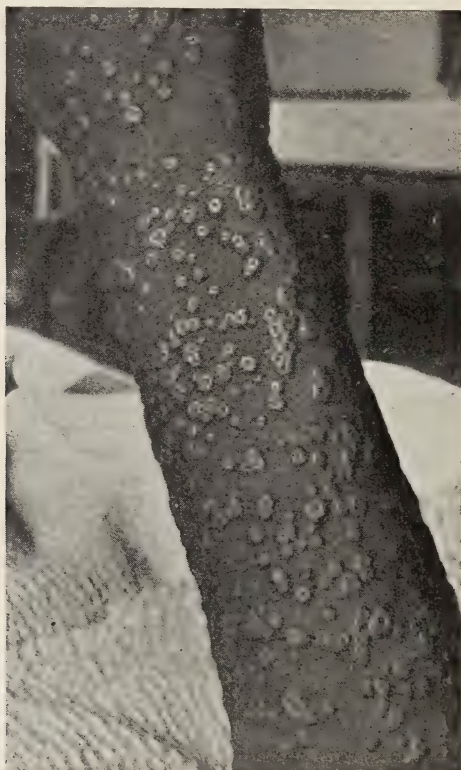
Fig. 88.—The eruption of small-pox, showing marked umbilication and confluence of the vesicles. From the collection of Dr. S. D. Hubbard.

height and the severity of the other symptoms depend directly upon the extent of the suppuration. The fever on the eighth or ninth day may reach 104° or 105° F. Over the face, scalp and hands the pustules are usually abundant and the swelling, burning and itching correspondingly intense. There are also sore throat, dysphagia, hoarseness, cough, etc., corresponding to the lesions of the mucous membranes. The temperature falls with the drying of the pustules and in moderate cases is normal before the end of the second week. With pustulation there is a well-marked leukocytosis.

The Confluent Form.—The stage of invasion does not differ from that

of the discrete form except in being rather more severe and often some-what shorter in duration. The rash appears on the third or fourth day, and is very profuse. The papules closely stud the skin of the face, scalp and extremities, and as they change into pustules many of them coalesce and form large suppurating areas (see Fig. 88). The face is greatly distorted by the swelling and hyperemia, the eyes are closed by edema, the suppurating areas emit a sickening odor and the pain, burning and itching are often intolerable. Over the trunk the pustules always remain discrete. The formation of numerous pustules and ulcers in the mucous membranes adds to the distress of the patient. The laryngeal symptoms in particular are apt to be severe. The cervical lymph-nodes are greatly swollen and suppuration is not uncommon. There may be diarrhea.

Corresponding to the extension of the suppuration the secondary fever is high, prolonged, and often irregular and septic in character. Delirium is common, prostration marked, the pulse grows progressively weaker and many of the patients die of exhaustion or of a complicating bronchopneumonia. In those that recover convalescence is slow and the cicatrization and pitting extensive.

The Hemorrhagic or Malignant Form.—In this type the constitutional symptoms from the beginning are especially severe. The rash is atypical and often scanty and the patient may die before it is well developed. On the second or third day there appear petechiæ and larger hemorrhages into the skin and conjunctiva, followed by bleeding from the mucous membranes of the nose, mouth, stomach, intestines, kidneys, bronchi, uterus, etc. Extensive extravasation of the blood beneath the skin may frightfully disfigure the face and body and justify the name '' black small-pox.'' The victim usually dies between the third and the sixth day. In some cases hemorrhages are not seen until bleeding occurs into the fully formed vesicles and pustules.

Varioloid is the name given to the mild form of small-pox which occasionally develops in persons who have been vaccinated. The initial stage is less severe than in true variola and with the appearance of the papules, on the third or fourth day, the temperature becomes normal and does not again rise. The eruption is scanty and is confined chiefly to the face, scalp and hands. The scattered pustules heal rapidly and there is little or no scarring. The patient may at no time feel ill enough to go to bed, but it is important to remember that from such a mild case the most virulent type of small-pox may be contracted.

Complications.—Severe laryngitis and bronchopneumonia are the most important complications. The former may excite a fatal edema of the glottis, or may result in necrosis of the cartilages. There may be pleurisy. Heart complications are rare. Parotitis, glossitis, membranous angina and otitis media are met with. Actual nephritis is not common. During the pustular stage, pyemia, septic arthritis and phlegmons may occur.

Severe conjunctivitis is common and may lead to keratitis and corneal ulceration.

During convalescence boils and abscesses often occur, and both local and multiple neuritis are met with. Inflammation of the substance of the brain or cord may give rise to hemiplegia, aphasia, paraplegia, etc.

Diagnosis.—Variola cannot be positively identified before the appearance of the eruption; but when the disease is prevalent every case of illness beginning abruptly with a chill and with intense headache and lumbar pain should be at once isolated. The initial rashes may in children be mistaken for those of measles or scarlet fever. Causes of hemorrhagic small-pox present many difficulties. They may be confused with other purpuric conditions or may be fatal so early that only a most careful search will disclose the beginning eruption. In the stage of eruption the disease is usually easily recognized. In varicella the eruption is most abundant upon the trunk; the papules have not the shot-like induration; the vesicles are very superficial and are usually not umbilicated; there is no surrounding zone of hyperemia; and few of the spots go on to pustulation. All stages of the eruption may be seen at one time. Moreover, the onset is usually not severe; the fever is slight, and the eruption appears upon the first or second day. The pustular eruption of syphilis may resemble that of small-pox, but the violent onset and the constitutional symptoms are lacking, and the distribution is different.

Prognosis.—The mortality among unvaccinated persons varies in different epidemics between 25 and 40 per cent. The death rate is highest in young children, drunkards and pregnant women. Hemorrhagic small-pox is regularly fatal, and the severe, confluent form is so in a majority of the cases. On the other hand, in varioloid (*i.e.*, among the vaccinated) the death rate is not more than 1 or 2 per cent.

Treatment.—Prophylaxis.—Every person who has been exposed to infection should be immediately vaccinated, as immunity can even then be acquired in time to prevent the attack. Whether vaccination after the disease has actually begun can mitigate its severity is, however, doubtful.

The patient should be rigidly isolated (best in a special hospital) and should remain so until all the crusts and scabs have disappeared. The greatest care is to be exercised in the disinfection of the bed linen, clothes, discharges, etc. (see Typhoid Fever), and in the proper disinfection of the sick-room and its contents after convalescence.

The treatment of the patient consists in very careful nursing and in meeting the symptoms as they arise. The diet should be fluid during the period of fever. The severe pain at the onset will often require morphine. The fever and the severe nervous symptoms are best met by cool baths or wet packs. The care of the eyes is very important—they should be kept clean and free of secretion by being bathed several times a day with a saturated solution of boracic acid.

In the stage of pustulation the intense burning and itching of the face and hands may be somewhat relieved by the constant application of cold compresses wet with a solution of carbolic acid 1–100. Whether pitting be extensive or not will depend upon the depth of the pustules, and

none of the many methods suggested for its prevention will avail in the severe confluent forms. As the pustules begin to dry the crusts should be kept moist or softened with vaseline or glycerine, to prevent the dissemination of the contagium. Nothing is known that will control the profuse hemorrhages of the malignant form.

VACCINIA—VACCINATION

Definition.—Vaccinia or cow-pox is an infectious disease of cows, closely allied to small-pox, and marked by a similar eruption, material from which, when inoculated into man, produces a local infection and is followed by more or less complete immunity from the latter disease. The means of protection against small-pox (vaccination) was discovered by Jenner in 1796, and by its almost universal use has changed variola from the commonest and most dreaded of the great pestilences to an infrequent and relatively harmless disease.

Bacteriology of Vaccinia.—In spite of a vast amount of investigation, the active agent in vaccinia, as in small-pox, is entirely unknown. The vaccine lymph contains various micro-organisms, but none of them has yet been proved to be the cause of the disease. The same intracellular bodies described by some as the specific organism of small-pox (cytorrhyctes variolæ) have been found in the lesions of vaccinia, but, as in small-pox, their significance is questionable.

Vaccination.—The virus or vaccine is obtained either directly from the vesicles of the calf (animal lymph) or from the vesicle of a vaccinated person (humanized lymph). The former method is much to be preferred. The lymph is now almost altogether supplied from carefully managed vaccine farms where great care is taken to have it pure and free from contamination. Vaccine lymph is furnished either dried on small bone points or quills, or preserved in glycerine in sealed capillary tubes.

Vaccination is done by carefully cleansing the skin with alcohol, scarifying an area ⅛ inch in diameter with a sterile needle or lancet and then well rubbing in the lymph and allowing it to dry before the clothes are adjusted. The spot should be kept clean and should be protected by a simple dressing or shield, to prevent contamination from scratching. The outer surface of the left arm, at the point of insertion of the deltoid tendon, is the most convenient place for vaccination.

Symptoms.—On the third day after vaccination a small red papule appears at the site of inoculation. This increases in size and on the fifth or sixth day becomes a vesicle. By the eighth day the vesicle is fully developed, distinctly umbilicated, and the edge is indurated and reddened. By the tenth day the vesicle has become a pustule which is surrounded by a considerable zone of inflammation and hyperemia. In two or three days this begins to subside and by the fourteenth or fifteenth day the pustule has become a thick, firm scab, which in the course of a week or ten days drops off and leaves a rounded, depressed scar. As the

vesicle develops there is apt to be much burning and itching and sometimes for several days a moderate fever. The axillary lymph-nodes on that side are usually enlarged and tender.

COMPLICATIONS are rare and arise almost always from lack of cleanliness or from a debilitated state of health. Cellulitis, sloughing, axillary abscess and erysipelas are all occasionally met with. A few instances of the transmission of syphilis by the use of humanized vaccine are known. Tetanus is another very rare complication. Erythema multiforme, urticaria, purpura, and other rashes are sometimes seen. It happens rarely that vaccination is followed not only by a local pustule, but by scattered pustules on various parts of the body, generalized vaccination.

The IMMUNITY from small-pox furnished by vaccination varies much, both in its duration and in its completeness. In most persons there is complete protection for a number of years, but eventually the immunity lessens and if exposure occur, small-pox in its modified form (varioloid) may develop. Oftentimes, however, immunity lasts throughout life.

Every child should be vaccinated when a few months old and should be revaccinated at intervals of eight or ten years and whenever an epidemic prevails. Persons who have been exposed to small-pox, if they be vaccinated immediately, can usually acquire immunity in time to prevent an attack.

VARICELLA

(Chicken-pox)

Definition.—Varicella is an acute, contagious disease of childhood marked by a discrete, vesicular eruption and by mild constitutional symptoms. It has no relationship to small-pox.

Etiology.—The disease occurs sporadically and in epidemics. It is contagious from the onset to the falling off of the crusts. Its specific germ is not known. Children between the ages of two. and ten years are chiefly affected. Immunity is usually procured by one attack.

Morbid Anatomy.—The only characteristic lesions are those of the cutaneous eruption.

Symptoms.—The PERIOD OF INCUBATION is quite regularly about two weeks.

The disease usually begins with malaise and slight fever, but often the eruption is the first thing noticed. This appears on the first or second day as small red papules upon the back, face and scalp, which gradually spread over the rest of the body. The papules are scanty and widely scattered, and each is surrounded by a small red areola. Within a day or two most of the papules have developed into large, clear, superficial vesicles which are not umbilicated and which in another day or two begin to dry at the center and to form crusts. In the meantime other crops of papules have appeared and it is characteristic of the disease to find fresh papules, vesicles and crusts all present at the same time. The eruption is never confluent and only exceptionally do the vesicles go on to

pustulation. In the course of a week or two the crusts separate and fall off and as a rule leave no scar. A few vesicles are often seen in the mucous membrane of the mouth and occasionally in that of the larynx. The slight fever and other constitutional symptoms usually disappear in two or three days. Occasionally the temperature reaches 103° or 104° F. During the eruption the itching is often very troublesome, but otherwise the children usually feel quite well. Rarely the eruption may be so severe and profuse as to threaten life. Erysipelas, nephritis, suppurative adenitis and gangrene of the skin are rare complications. The prognosis is almost invariably good.

Diagnosis.—The mild onset, the early appearance of the rash, its scantiness, the absence of the shotty induration, of umbilication, and of pustulation, and especially the presence at the same time of all the stages of the eruption, serve to distinguish the disease from small-pox. Usually, too, there is a history of exposure.

Treatment.—Isolation is not necessary unless there be other small children in the household. If instituted, it must be maintained until the skin is entirely clear of scales (about three weeks). Other children in the family should be kept from school for the same period. No treatment is needed as a rule, except the application of carbolated vaseline or some bland ointment to the skin to allay the itching. In young children care must be taken to prevent scratching.

TRYPANOSOMIASIS

Definition.—The morbid conditions caused by infection with trypanosomes, including protracted irregular fever, localized erythema or edema, anemia and emaciation, and terminating in the sleeping sickness.

Etiology.—Within the last ten years infection of man with trypanosomes has been recognized. The parasites had been long known in the blood of horses and other mammals. Human infection has been found prevalent throughout the Congo basin and generally in Central Africa. The distribution of the disease is found to correspond to that of the tse-tse fly, which is now regarded as the intermediary host of the parasite. Europeans are rarely affected, while the native population is very susceptible.

TRYPANOSOMES. — The parasite found in human blood, known as trypanosoma gambiensis, is an organism with a long, spindle-shaped body, rounded at one end, terminating at the other in a long flagellum, the continuation of a delicate undulating membrane which fringes the spindle-shaped body. The body contains a nucleus and several masses of pigment. The parasites have been found in the blood, cerebrospinal fluid, and lymphatic nodes. They are stained by the Romanowsky method. They are most easily found in the glands. In the blood they are few in number and are found only after centrifugation and very careful examination. In the cerebrospinal fluid they appear only in the advanced stage of the disease known as sleeping sickness.

Thus far artificial cultivation of the human trypanosome has not succeeded, although the kindred organisms found in the rat and horse have been grown on suitable media.

TRANSMISSION.—The infection is spread by the bite of the tse-tse fly, the organism being carried from the sick to the healthy. Whether the organism, like the malarial parasite, undergoes a certain development in the body of the intermediary host, or is merely carried by it, is not clearly established. By reason of the prevalence of the tse-tse flies along the shores of lakes and streams these regions are peculiarly dangerous.

Morbid Anatomy.—The gross changes are those of anemia and emaciation. The spleen and liver may be enlarged, probably from previous malaria. A lymphocytic infiltration of the pia-arachnoid following the blood-vesels into the brain and cord is found microscopically. Similar perivascular infiltration is found throughout the body.

Symptoms.—The PERIOD OF INCUBATION is about 20 days in experimental inoculation in monkeys; its exact duration in man is unknown. The onset is slow and insidious; months or even years may elapse before definite symptoms develop. Enlargement of the lymphatic nodes is an early sign, and there may be no other. Fever of variable height and duration occurs in most cases, with progressive anemia and exhaustion.

SLEEPING SICKNESS.—Sooner or later the final phase of the disease is reached. Progressive feebleness, apathy, lethargy and an unconquerable tendency to sleep terminate finally in coma and death. During this stage an irregular fever is present, headache and indefinite pains are complained of, tremor of hands or tongue may be noted, bed sores may develop. Patchy erythema or edema is sometimes seen. The blood may show a slight leukocytosis with lymphocytosis and secondary anemia. Trypanosomes are found on careful examination.

Prognosis.—Recovery may occur in the early stage, but the sleeping sickness is regularly fatal. Towns have been wiped out and whole districts of Africa depopulated by this scourge.

Treatment.—PROPHYLAXIS.—The efforts at prevention follow the lines employed in relation to malaria. Isolation and protection of the infected persons to prevent infection of the flies cannot be satisfactorily practiced because the infection is so wide-spread and so long latent. People entering infected districts should avoid the shores of lakes or streams which are the natural habitat of the tse-tse fly and should protect themselves against its bite by screening houses and using mosquito netting.

The most effective treatment of the disease consists in the intramuscular injection of a 10 to 20 per cent. solution of atoxyl, a meta-arsenic-anilin compound, to the amount of one grain every third day, the dose being gradually increased. Much larger doses have been employed, but often with serious results. Blindness due to optic atrophy has repeatedly resulted from excessive doses.

KALA–AZAR

(Tropical Splenomegaly, Dum-dum Fever, Black Sickness)

Definition.—A chronic infective disease showing the presence in the internal organs of certain parasites, the Leishman-Donovan bodies, and characterized by chronic irregular fever, marked enlargement of liver and spleen, progressive anemia, exhaustion and death.

Etiology.—The cause of the disease is infection with the Leishman-Donovan bodies, now regarded as forms of trypanosomes and given the name of herpetomonas Léger. Two forms of the parasite are known, one as it is found in the internal organs or blood of man, the other obtained by growth on culture media. In the first form the organism is a small oval or rounded body 2–4 micra in diameter, having two chromatin masses, one larger, the nucleus, and a smaller nucleolus. The parasites multiply by fission, the nucleus and nucleolus dividing before the body. They are regularly intracellular, being found especially in the endothelial cells of blood- and lymph-vessels, and most abundantly in the spleen and liver.

The cultural forms are much larger, 12 to 20 micra in length, and flagellated, but without the undulating membrane of the trypanosoma gambiensis.

It is assumed that the parasite is transmitted from man to man by the bite of some blood-sucking insect or tick, but as yet this is not proven. The disease is endemic in Southern Asia, especially India, and Northern and Southern Africa. Local epidemics are known.

Morbid Anatomy.—The spleen and liver are both enlarged, the spleen sometimes enormously. In the cells of both organs the parasites are found in large numbers. The liver has a brown or mottled section. The bone marrow also shows the parasites in great numbers. The lymphatic nodes are generally enlarged. Intestinal ulcers are common. The blood and all the viscera may show the parasites.

Symptoms.—The INCUBATION PERIOD is less than ten days. The onset is marked by high fever, possibly a chill, which continues from two to six weeks, and is accompanied by marked enlargement of liver and spleen. A period of apyrexia follows, to be succeeded by fever and splenic and hepatic swelling. These periods of fever and apyrexia continue indefinitely. Anemia, emaciation and finally edema of the legs or ascites appear. At the end of a year or two the patient dies of exhaustion or of intercurrent dysentery, pneumonia, or phthisis.

The blood shows a secondary anemia with a marked hypoleukocytosis; the ratio of red to white cells may be 2,000–4,000 to one. There is a relative lymphocytosis. The parasite may be demonstrated by Leishman's or Giemsa's stain.

Diagnosis.—Malaria must be excluded by repeated examination for the plasmodia and the trial of quinine. Other tropical fevers, such as trypanosomiasis, can be differentiated only by the demonstration of the parasites in the blood or in material obtained by puncture of liver or

following this procedure. The parasites have been demonstrated in smears from excised lymph-nodes (Cochran).

Treatment has, therefore, been unsatisfactory. Atoxyl may be given in 2-grain doses every third day. Change to a healthful climate, rest and good food are indicated.

OTHER PARASITIC PROTOZOA

A number of other varieties of protozoa have, at times, been found in the human body. (1) Cercomonas intestinalis, a pear-shaped flagellate, found in the feces. (2) Trichomonas intestinalis, a similar pear-shaped organism endowed with ameboid movement. (3) Balantidium coli, an ovoid body, 7 to 10 micra in length, provided with ciliæ, also found in the feces, especially in diarrheal conditions. (4) Trichomonas vaginalis, similar to the intestinal form, but smaller. Whether any of these varieties is of pathogenic importance has not been established.

DISEASES DUE TO ANIMAL PARASITES OTHER THAN PROTOZOA-METAZOA

The animal parasites of man other than protozoa are sometimes grouped under the broad title of metazoa. They include a number of diverse groups, some but distantly related to the others, and these groups are constantly being added to. The common metazoan parasites, however, are of these varieties.

(1) Trematoda (literally holed, or having holes), leaf-shaped organisms, having an intestine, but no anus, and one or two suckers; parasitic in the lungs, liver, intestine; blood flukes.

(2) Cestoda (the cestus, banded), long, segmented organisms, with suckers, but no intestine; adults, parasitic in the intestine, the larvæ elsewhere; tape-worms.

(3) Nematoda. (Thread-like organisms.) Intestine present; parasitic in intestines, muscles and lymphatics, including common round worms, filaria and trichinella spiralis.

DISEASES CAUSED BY TREMATODES
DISTOMATOSIS : FLUKE INFECTIONS

Definition.—Infection by a trematode. The parasite may lodge in the lungs, liver, intestine, or veins of any part of the body. The resulting condition is designated as pulmonary, hepatic, intestinal or venal distomatosis.

Etiology.—The life-cycle of the parasites is complicated, and may involve two or more generations living outside of man. The parasites are hermaphrodites. Direct infection from man to man appears possible.

The disease is almost unknown in the United States, but very common in Japan and China, whence practically all our knowledge of it is derived.

Morbid Anatomy.—In the lungs thick-walled cavities filled with muco-pus, blood and ova, and communicating with the bronchi, are formed. The parasites may be found in them.

Symptoms.—PULMONARY DISTOMATOSIS.—A chronic cough with an abundant brownish or reddish muco-purulent expectoration is the chief symptom. Blood is commonly present, and hemorrhages not infrequent.
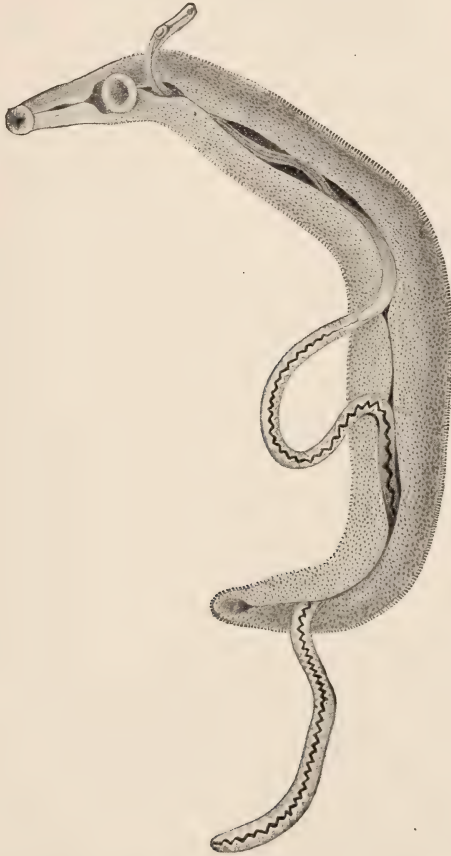
FIG 89.—Bilharzia hematobia, or Schistosomum hematobium, showing female lying within the male. (Copied from Braun.)

The condition suggests tuberculosis. The diagnosis is made by finding the characteristic ova in the unstained sputum.

PARASITE.—The paragonimus Westermani, as it is now called, is a plump oval or pyriform fluke, 8 to 16 mm. by 4 to 8 mm. and about 2 to 4 mm. in thickness.

The eggs are oval bodies, measuring 50 by 90 micra, with an operculum or lid at one extremity. They are usually found in numbers.

HEPATIC DISTOMATOSIS—LIVER FLUKE.—The parasite is known as fasciola hepatica, and is a small trematode, measuring from 20 to 30 mm. by

8 to 13 mm.   The eggs are yellowish-brown, oval bodies, 140 by 70 micra.
The liver is enlarged and tender; jaundice or ascites may develop.   The
ova are found in the stools.

Other liver flukes, bearing the name of opistorchis sinensis, have been
described.

VENAL DISTOMATOSIS.   BLOOD FLUKE—BILHARZIA HEMATOBIA.—In this
condition the flukes lodge in the portal vein, or the veins of the intestine
or bladder wall.   Infection is brought about through drinking contam-
inated water.   The disease is common in the tropical and sub-tropical
climates, especially in Egypt.   It has been observed in Cuba and Porto
Rico, and in a few instances in the United States.

Morbid Anatomy.—The ova, not the parasites themselves, are the
active agents in the disease.   From the portal or other veins where the
worm is lodged the ova make their way into the walls of the intestine,
bladder or rectum.   Here they are grouped in masses, showing as whitish
specks, surrounded by inflammatory thickening.   Ulcers may develop or
a diffuse thickening with papillary outgrowths.

Symptoms.—Two forms of the disease are recognized, according as
the bladder or rectum is most involved.   In the former hematuria is
present, possibly cystitis, pyelitis, or pyelonephrosis.   In the latter diar-
rhea with discharge of blood and pus.

Diagnosis—Microscopic examination reveals the presence of the char-
acteristic eggs in the urine or feces.   The ova are spindle-shaped with a
small spine projecting from one side or extremity.   They measure 0.12
to 0.19 mm. by 0.05 to 0.07 mm.   Within the egg is a ciliated embryo
which is set free when the eggs are placed in water and thus becomes
the means of spreading infection.

THE PARASITES.—The male Bilharzia is 4 to 15 mm. long by 1 mm.
in breadth (see Fig. 89).   The female is filiform, from 15 to 20 mm. in
length and lies with a canal in the body of the male.

Treatment.—Male-fern or santonin may be given, but with doubtful
effect.   The treatment must be supportive and symptomatic.

DISEASES CAUSED BY NEMATODES

ASCARIASIS—ROUND-WORM OR EEL-WORM INFECTION

Parasite.—Ascaris lumbricoides resembles the common earth-worm
(angle-worm), but is longer and more pointed at the extremities.   The
male measures from 15 to 20 cm. in length, the female from 20 to 40 cm.,
the diameter varying from 3 to 5 mm. (see Fig. 90).   Their habitat is
the small intestine.   The eggs develop into embryos which grow into the
adult forms.   No intermediate host is required (see Fig. 91).

Etiology.—Infection with the round-worm is the most frequent par-
asitic infection of man.   Children most often harbor these worms, but
they may be found at any age.   The infection is carried by contaminated
water or food.   The house-fly may be the means of spreading contagion,
particularly in country districts where privies are used.

Symptoms.—In the great majority of cases no symptoms are excited. In other cases, especially if many worms are present, all manner of disturbances of the stomach, intestine, and nervous system may be produced. Loss of appetite, nausea, or vomiting, colicky pains, diarrhea or constipation, headache, vertigo, convulsions, chorea, epilepsy and the like have all been attributed to round-worm infection. Particular significance is commonly attributed to fretfulness and irritability, picking at the nose, dark rings under the eyes, grinding of the teeth, or offensive breath. Most probably these symptoms are due to improper feeding rather than to the presence of the round worms.

FIG. 90.—Ascaris lumbricoides. $\frac{1}{3}$ to $\frac{1}{6}$ natural size.

The most pronounced symptoms of ascariasis are due to the habit of the worm of wandering from the small intestine into other parts. They may block the bile-ducts and produce jaundice; crawling into the stomach they may excite persistent vomiting and be ejected per orem; they may block the larynx and cause death by suffocation; or they may pass through the Eustachian tube and escape through the ear.

Diagnosis.—The appearance of the worms in the feces, or in vomitus, etc., is conclusive. They are often seen in the stools, without symptoms of any kind. The eggs are easily recognized in feces. They are oval bodies, measuring 50 to 70 micra in length and 40 to 50 micra in breadth, with a thick transparent shell usually covered by an albuminous coat. The body of the egg is unsegmented.
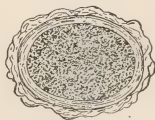
FIG. 91.—Ovum of ascaris lumbricoides, showing shell and envelope. × 400.

Prophylaxis.—The essential points are the proper disposal of fecal material, and personal cleanliness. In cities in this country ascaris infections are becoming infrequent except in the tenement house population.

Treatment.—Santonin, usually combined with an equal amount of calomel, is given in divided doses, one-half to one grain every half hour to a total of from 2 to 5 grains for an adult. The dose should be repeated from time to time so long as ova appear in the feces.

OXYURIASIS—PIN-WORM INFECTION

Parasite.—Oxyuris vermicularis, the pin-worm, seat-worm or threadworm, is a small, white nematode, from 3 to 5 mm. (male) to 10 mm. (female) in length. The adult worms inhabit the small intestine, where they copulate. The fertilized female wanders to the cecum or colon.

30

Eggs are passed, and often the female worms in numbers wander from the anus, and are found in the skin, in the vagina or bladder.

Etiology.—As the female worms containing ova wander from the anus and the eggs are also passed in the feces, infection may be direct or by means of contaminated water or vegetables. Autoinfection is doubtless frequent and serves to perpetuate the condition.

Oxyuris infections are extremely common in children, rare in adults.

Symptoms.—Inflammation of the anus caused by scratching to relieve the itching produced by the presence of the parasites, restlessness and sleeplessness, since the itching is worse at night, are characteristic. All or any of the indefinite symptoms narrated under ascariasis may be attributed to the oxyuris.

In children the oxyuris frequently lodges in the appendix and may be a cause of appendicitis (Cecil and Bulkley).

Diagnosis.—The worms, looking exactly like active little white threads, can often be seen in the feces, in the creases of the anus or on
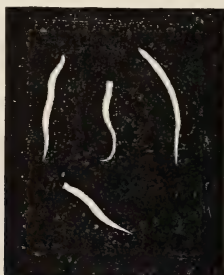
Fig. 92.—Oxyuris vermicularis. Natural size.

the adjacent parts (see Fig. 92). Eggs may be found on microscopic examination of the feces. The eggs are oval, measuring 50 by 20 micra, and embryos can often be seen in them, distinguishing them from the unsegmented ova of ascaris lumbricoides.

Treatment.—Santonin and calomel, as for ascaris, should be given to remove the young worms from the small intestine. Infusion of gentian, thymol or betanaphthol may be used for the same purpose.

To clear the colon and rectum, injections of various kinds are used. Infusion of quassia, lime water, or strong salt water or salt in milk may be used. Weak carbolic acid and bichloride of mercury solutions have been used, but the possibility of poisoning should forbid their employment. The enemata should be repeated daily for several times, then at intervals till all signs of infection have disappeared. Both the parasites and ova should be sought for.

TRICHINIASIS

Parasite.—Three stages of the parasite, trichinella spiralis, are to be distinguished: (1) The adult worms living in the small intestine; the males 1.4 to 1.6 mm. in length, the females 3.0 to 4.0 mm. The males

die shortly after copulation. The females remain in the intestine or bore into its lymphatic spaces, and being viviparous, there deposit their young, about 1,500 for each female worm. (2) The embryos, microscopic, thread-like organisms, grow to 100 micra in length, pass into the blood stream from the lymph spaces, and are thus distributed throughout the body. The embryos reach the muscles in from 10 days to 2 weeks following infection. (3) Having reached the muscles the embryos coil themselves up into round or oval bodies and become surrounded by a fibrous capsule, thus forming the encysted larvæ, the infecting stage of the parasite. When meat containing such larvæ (the so-called measles) is eaten, the capsule is dissolved off, the embryo set free in the intestine, and the chain of reproduction renewed.
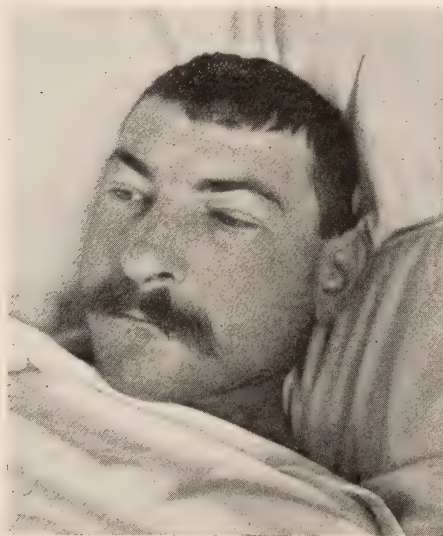
Fig. 93.—Patient with trichiniasis, showing the puffiness of the eyelids.

The larvæ may be found in the meat of a number of animals, such as rats, dogs and cats, but for man the pig is the source of infection. Hogs are infected through eating the flesh of rats containing the larvæ, and the rats are probably infected from other rats or from the refuse of abattoirs.

The larvæ are killed by thorough cooking, so that the disease is common only among people who, like the Germans or Australians, habitually eat raw pork. It regularly occurs in localized outbreaks, when the meat of a trichinous hog has been consumed by a family or group of persons. Whole villages have been poisoned in this way. Germany now maintains a costly microscopic inspection of all hogs slaughtered in her abattoirs in the effort to prevent such occurrences.

In America the disease is met with in sporadic cases, occasionally in

the several members of one family. The incidence and severity of the disease seem to depend on the amount of the infected pork eaten.

The disease becomes more frequent in this country as the number of people accustomed to eating raw pork increases.

Symptoms.—The PERIOD OF INCUBATION is usually from 10 days to 2 weeks. Diarrhea with abdominal pains may occur during this period, but is usually absent. The invasion of the disease corresponds to the lodgment of the embryos in the muscles of the victim. A puffiness (edema) of the eyelids and face may be noted at the outset (see Fig. 93). Then follow fever, low or high, pains in the muscles, sometimes exquisite tenderness, especially in the arms and legs, profuse sweats, loss of appetite, nausea or vomiting, constipation and headache.

Except for the edema of the face, muscular pains and tenderness, the onset closely resembles typhoid fever. All these symptoms continue for
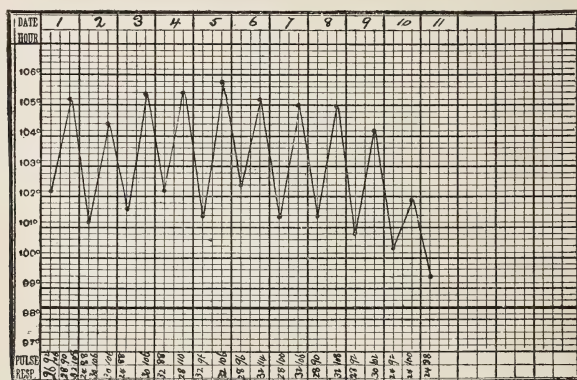
FIG. 94.—Typhoid-like fever of trichinosis.

from 2 to 7 weeks, according to the severity of the infection. The patients may die of exhaustion, but for the most part recover slowly.

The fever of trichinosis is of the continuous type with remissions usually more marked than those of typhoid (see Fig. 94). It may be high or low. It falls by lysis. The spleen is enlarged and in some instances there is a roseola which cannot be distinguished from the rash of typhoid fever. Headache and mental dulness are usually pronounced. The muscle pains and tenderness are often absent or very mild.

BLOOD.—A leukocytosis of 15,000 or more is present in most cases and the differential count shows an eosinophilia varying from 5 to 50 per cent., or even more.

Diagnosis.—The resemblance to typhoid fever is very close. The puffiness of the eyelids and face is very suggestive. The leukocytosis and eosinophilia are almost conclusive. The final proof lies in finding the embryos either in the blood or muscle. T. C. Janeway has shown that

the embryos may be found in the blood by the method experimentally developed by Staübli. Several cubic centimeters of the blood (2–10) are drawn from a vein by a hypodermic needle, laked with from 10 to 15 parts of 3 per cent. acetic acid, centrifuged, and the embryos sought in the sediment. It is not yet known how reliable the method is in man. If it fails, a fragment of any muscle, the deltoid, biceps, or gastrocnemius, for example, may be excised under cocaine, hardened, sectioned, stained and examined microscopically. A characteristic myositis with many eosinophile cells is found here and there, and in some of the areas the encysted larva will be discovered. If few in number, it may require the examination of many sections to demonstrate the larvæ.

Prognosis.—The disease is fatal in about 5 per cent. of cases. The milder cases regularly recover.

Prophylaxis.—Thorough cooking of all pork or pork products will kill the parasites. Microscopic examination of the flesh of all slaughtered hogs and the rejection of all infected carcasses have greatly reduced the prevalence of the disease in Germany, but many local outbreaks and sporadic cases still develop, because the meat is often eaten raw.

Treatment.—By the time the disease comes under observation the embryos have already left the intestine and are lodged in the muscles and other tissues. Purgatives and anthelmintics, therefore, are of no avail. The treatment must be wholly symptomatic and supportive. Hot baths or morphine may be used to relieve severe muscular pains. Atropine will check the profuse sweating.

UNCINARIASIS OR HOOK-WORM DISEASE

Parasite.—Two distinct species of uncinaria are known, an Old World and a New World hook-worm. The Old World form, the ankylostomum duodenale, or uncinaria duodenalis, is commonly found in Europe, Asia, Africa and Australia. It is but 8 to 18 mm. in length. The head is armed with a number of sharp teeth, by which it fixes itself in the intestinal mucous membrane. The species found in this country, uncinaria americana (Stiles), is shorter, 7 to 11 mm., and presents certain differences in the minute anatomy of the head (see Fig. 95).

The eggs of the two species are oval, measuring 60 or 70 micra by 40 (see Fig. 96). They are usually somewhat segmented and may contain fully developed embryos. No intermediary host is required.

Both parasites inhabit the small intestine, more often jejunum or ileum than duodenum.

Etiology.—The eggs of uncinaria passed in the feces (see Fig. 96) undergo development in moist earth or water. Infection may take place by one of two routes.

(a) By ingestion of the ova or embryos in contaminated water or food.

(b) By passage of the embryos through the skin into the circulation, and thus to the lungs, then up the air tubes to the pharynx, and so into the stomach and intestine. Roundabout as this route is, it has been

demonstrated by Loos and others, and infection through the skin doubtless plays a considerable part among tropical people wearing no shoes and in constant contact with the soil.

Uncinariasis has long been known throughout the tropics, and in parts of Europe, especially Italy and Switzerland. Recently it has been shown to be very prevalent in the Southern States, Cuba and Porto Rico. Ninety per cent. of the inhabitants of Port Rico and of certain districts in the South harbor these parasites. Children suffer particularly. Rural communities, farmers, brick-makers, tunnel workers and others in close contact with the soil are especially liable. The infection is rare in the Northern States, and particularly in cities.

Symptoms.—(1) There may be no symptoms, and the eggs may be found in casual examination of the feces.
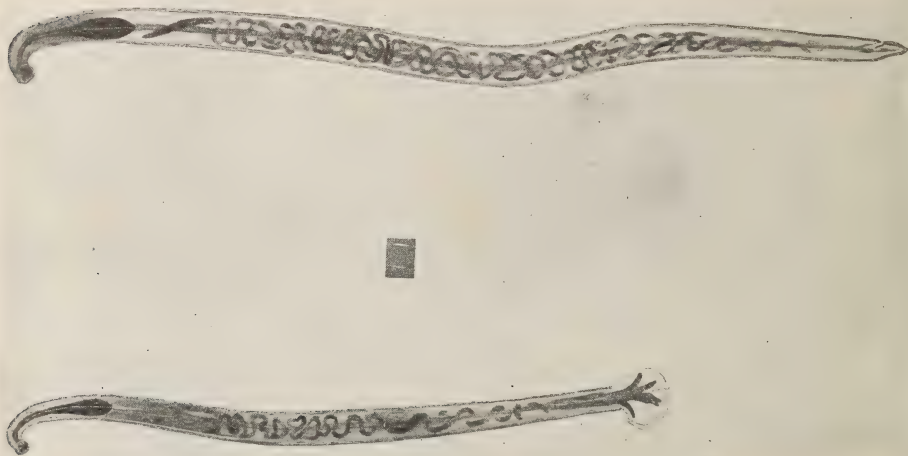
Fig. 95.—Uncinaria americana. Upper figure, female × 63; lower figure, male × 63. The white lines on the dark square show the natural size. (International Clinics, Vol. IV, Series 20, page 142.)

(2) Anemia, with pallor, palpitation, etc., marks the majority of cases. In some instances the anemia is profound, and the blood picture is that of pernicious anemia.

(3) The dirt-eaters of the South, with their stunted growth, anemia, emaciation, protuberant abdomens, edema, or ascites, and physical or mental lethargy, are types of severe chronic infection with the ankylostomum.

THE BLOOD.—Anemia, varying from a mild secondary anemia to typical pernicious anemia, is present. Leukocytosis is unusual. Eosinophilia is common, but may be absent in chronic cases.

Prognosis.—The infection in its milder forms is curable. The severer cases are often fatal. Thirty per cent. of the deaths in Porto Rico have been attributed to uncinariasis. The prevalence of the infection in a considerable portion of the poorer population of the Southern States,

Porto Rico, Cuba and tropical countries renders it of vast economic importance.

Diagnosis.—In badly infected districts severe anemia or the clinical picture of dirt-eating are suggestive. In all cases of severe anemia, the feces should be examined for the ova, or after an anthelmintic for the worms, which are often present in numbers. (See Fig. 97.)

Prophylaxis.—The proper disposal of fecal material is the essential measure. In cities an adequate sewage system, in the country proper privies, in mines the pail system should be provided.

Treatment.—Thymol or male-fern is administered after the preparatory measures given under tape-worm infection. Thymol is given in 2
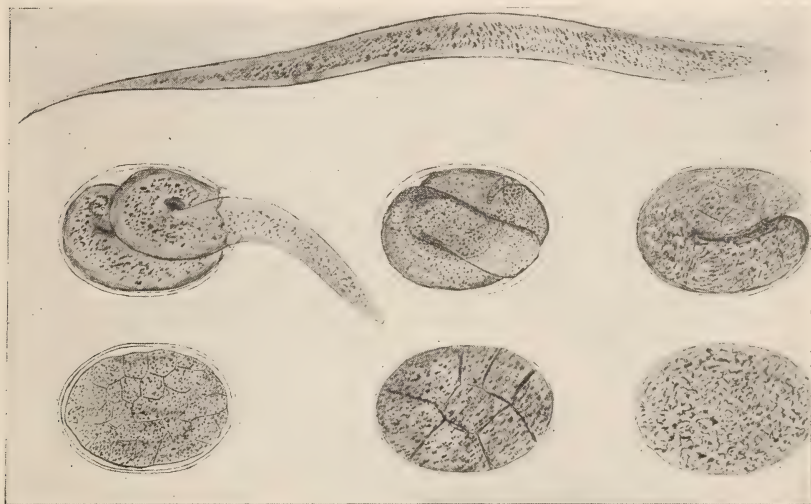
Fig. 96.—Uncinaria americana. Ova and larva magnified about 1500 times. (International Clinics, Vol. III, Series 20, page 143.)

dram doses in capsules, the dose repeated in two hours, and followed by a purge. Male-fern may be used as for tape-worm.

FILARIASIS

A considerable number of filaria have been found in man during recent years, but of these only two are sufficiently common to make them of practical importance: the filaria Bancrofti and filaria loa.

FILARIA BANCROFTI

The parent worms are delicate, transparent, hair-like organisms, transversely striated, 50 to 100 mm. in length. Two sexes are recognized and are regularly found together, often closely intertwined, in lymphatic varices or in lymph-nodes. They are viviparous. The larvæ, 300 to 400 micra in length, are found in the circulating blood. (See Fig. 98.) They are provided with a sheath and a sharply pointed tail. They appear in the peripheral circulation during the night, but by day crowd into the

lungs, heart and internal organs. If the patient sleeps by day and is about at night, the periodicity of the organisms is reversed and they appear in the peripheral blood by day. The larvæ are harmless. According to Manson, the symptoms of filariasis are produced by the blocking of lymphatics by the adult worms or the immature products of conception, whatever these may be.

The filarial larvæ drawn into the stomach of a mosquito undergo a certain development in the muscles of that insect and then make their way to the proboscis, after 20 or 30 days, and thence again reach the body of man and become mature parasites. The larval forms can survive for a time in water, and contaminated water may, therefore, be a means of infection.
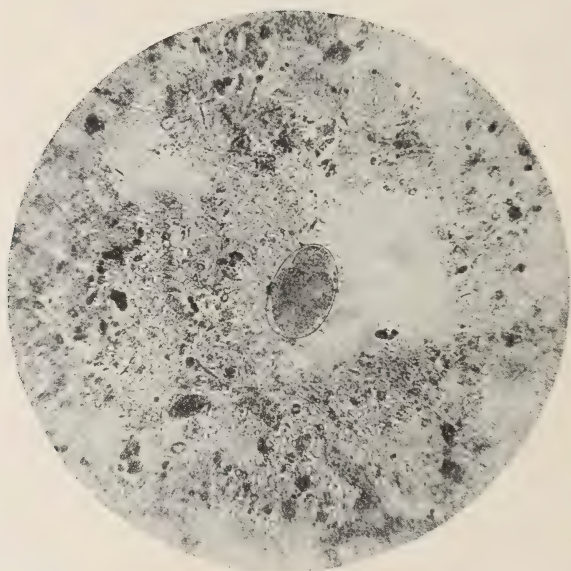
FIG. 97.—A photomicrograph showing the ovum of Ankylostomum duodenale in the feces. × 500. From the collection of Dr. Walter B. James.

These parasites are found in all tropical and sub-tropical countries, especially Africa, as far north as Spain and Charleston, S. C., and as far south as Australia.

Symptoms.—These depend upon blocking of lymphatics or lymphnodes by the parasites or their products and include lymphangitis, lymphatic varices, enlargements of lymph-nodes, abscesses, chylous effusions into the peritoneum or tunica vaginalis, chyluria, and elephantiasis. Of these, chyluria and elephantiasis are most important.

CHYLURIA.—This condition results from the rupture of lymphatic varices in the walls of the bladder or some other part of the urinary tract. The urine becomes milky white or pinkish, if blood as well as chyle is present (hematochyluria).

On standing the urine separates into three strata, an upper creamy layer, a reddish sediment, and an intervening milky fluid, usually containing a coagulum. The sediment shows red blood-cells, lymphocytes, granular fatty matter, epithelium, and usually filariæ. The middle and upper layers contain much granular fatty matter, and larger oil globules. The fat may be dissolved out by ether.

Chyluria comes and goes for months or years; when protracted, debility and anemia are produced. Retention of urine may result from stoppage by blood clots.

ELEPHANTIASIS.—Brawny thickening of the skin and subcutaneous

FIG. 98.—Filaria Bancrofti in the blood. From the collection of Dr. Walter B. James.

tissues due to lymphatic obstruction (elephantiasis) is now known to be a late consequence of filarial infection. The lower extremities, the scrotum, or penis are most often involved. The enlargement of the feet and legs may be enormous, or the scrotum and penis may form huge tumors. Attacks of lymphangitis, dermatitis and cellulitis, accompanied by fever (elephantoid fever), occur from time to time. With each attack the enlargement of the affected parts increases. The skin and subcutaneous tissues become greatly thickened, dense, and do not pit on pressure. All inflammatory symptoms subside, but the patients suffer from the weight and hindrance of the enlargement or tumors. Scrotal tumors of 10 to 15 pounds are common, and one of 224 pounds

has been reported. The enlargements are permanent. Filaria cannot be found in the tissues, but there are many reasons for believing filaria to be the cause.

Prophylaxis consists in protection against mosquitoes.

Treatment.—For chyluria rest with elevation of the hips, to lessen pressure on the broken lymphatics, is the only measure of value. For elephantiasis elastic bandaging, elevation and massage may be tried. Surgical removal of large tumors may be required.

FILARIA LOA

This thread worm closely resembles filaria Bancrofti. The adult male measures from 30 to 34 mm. in length, the female somewhat less. They are both marked by numerous rounded, translucent bosses on the cuticle. They are found in the subcutaneous tissue of any part of the body, especially of the extremities or of the eye. The microfilariæ can hardly be distinguished from those of f. Bancrofti, except that they are found in the blood by day, and are disposed in rather uncouth lines instead of the graceful loops or circles assumed by the microfilaria B. Manson says that one suggests the awkward, angular writing of a schoolboy, the other the easy flowing strokes of a skilled penman.

F. loa is widely distributed through tropical West Africa; it has rarely been seen in the United States.

Symptoms.—F. loa may cause pricking, itching or pain and irritation with transient local edema of any part of the body. It seems to prefer the loose connective tissue of the conjunctiva or eyelid and there may cause irritation and congestion.

Treatment.—F. loa has often been removed from the eye by incision and traction.

DRACONTIASIS

Parasite.—Dracunculus medinensis or filaria dracuncula, Guineaworm, is a remarkable parasite found in Southern Asia, tropical Africa and some parts of Brazil. It is found in many of the lower animals.

Nothing definite is known of the male worm. The female attains a length varying from 30 cm. to 120 cm., and a diameter of 1.5 mm. The body is cylindrical and milky white. It is mostly occupied by the uterus filled with coiled-up embryos.

The impregnated female is found in the connective tissue, nearly always in the lower extremities, but possibly in the arms or head. Boring downward she bursts through the skin, producing a small ulcer on the leg, from which milky fluid is discharged. On examination the fluid is found full of active embryos, measuring from 0.50 to 0.75 mm. in length.

Cyclops quadricornis, a minute fresh-water copepod or crustacean, is the intermediary host. The infection is, therefore, water-borne. The life-span of the female dracunculus is calculated at one year.

Symptoms.—Ulcers discharging a milky fluid are found at the point

of exit. If the parasite dies in the subcutaneous tissue an abscess may be caused, or the body of the worm may become calcified and be palpable as a cord beneath the skin.

Treatment.—When emptied of her embryos the worm is absorbed or can easily be extracted. Until that time, normally 15 to 20 days, extraction is difficult. Repeated douching of the ulcer with cold water causes contraction of the worm and hastens the evacuation of the embryos.

The parasite, if projecting, may be injected with bichloride of mercury, 1 to 1,000, which kills her, so that extraction can be practiced after 24 hours.

If the parasite does not project, but can be felt under the skin, she may be killed by injecting a few drops of the solution at several points about her, and then left to be absorbed or cut down upon and extracted.

DISEASES CAUSED BY CESTODES

Parasite.—Tape-worms are formed of a head (scolex) and detachable segments (proglottides) ; on the scolex form sucking disks by which it attaches itself, and in some cases a rostellum with hooklets. The segments may number hundreds, each containing a branched uterus from which ova in great numbers are discharged. Each ovum contains a minute embryo which under the conditions found in the intestines of various animals, cattle, pigs, fish (or even man) is set free, makes its way from the intestine to the muscles or viscera of the intermediary host and there lodges and becomes encapsulated, producing a cyst-like growth, variously called measles, cysticercus or hydatids. In this state they lie dormant indefinitely. When muscle or meat containing these cysts is ingested by man the embryo is set free in the intestine, effects lodgment and grows into the adult worm. Thus the common intestinal infection is produced. In certain rare instances man becomes the intermediary host with resulting infection with the bladder forms, cysticercus or hydatid disease.

INTESTINAL INFECTIONS

A considerable variety of tape-worms has been found in the human intestine, but of the number only four are common.

(1) Tenia mediocanellata (see Fig. 99, *A, B, C*). The beef tapeworm. Cattle are the intermediary hosts and man is infected by eating raw meat containing the embryos. This is the form most often found in the United States. The average length of the worm is 4 to 10 meters, though some are much longer. The head is roughly cubical and measures 1 to 2 mm. in diameter. It has a rostellum but no hooks. The uterus, which forms the conspicuous feature of each segment, sends out from 20 to 25 branches, each of which breaks up into a number of smaller tubes. The many divisions of the uterus constitute the chief differential of the segments from those of t. solium.

The eggs are spherical in form, 30 to 40 micra in diameter, with a distinct shell.

(2) Tenia solium (see Fig. 100, *A, B, C*). The pork tape-worm. The pig is the intermediary host, and man is infected by eating raw or poorly cooked pork. This worm is, therefore, common in Europe, but is very infrequently seen in the United States, unless in immigrants. It has an average length of 2 or 3 meters. The head is rounded, about 1 mm. in diameter, armed with a rostellum and a double circle of hooks. The ripe segments are very like those of tenia saginata, but the lateral branches of the uterus number only 10 to 12 and its finer divisions are not so numerous. The spherical eggs so closely resemble those of t. saginata that they cannot be distinguished microscopically.
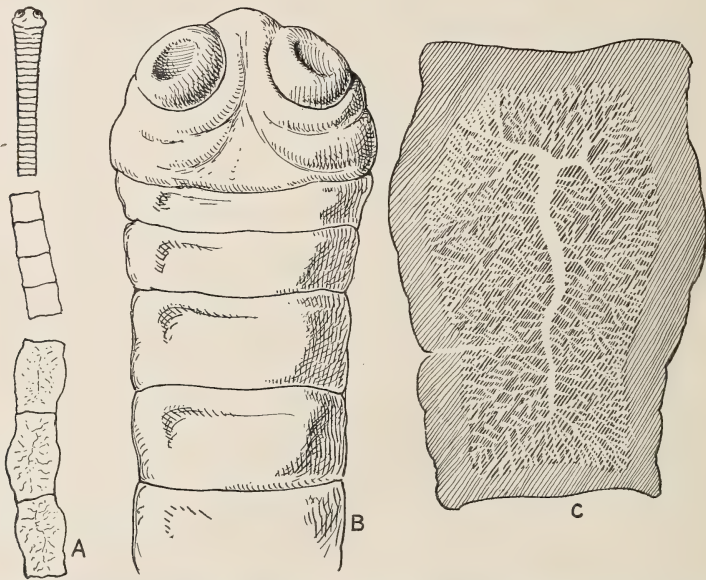
Fig. 99.—Tenia mediocanellata or saginata. *A*, head and segments, natural size; *B*, head, showing sucking disks but no hooklets, × 7; *C*, proglottis, showing branched uterus and sexual pore, × 7.

(3) Dibothriocephalus latus. Fish tape-worm. Fish are the intermediary hosts, especially pike, perch or salmon. It is, therefore, common in lake regions. It is frequently met with throughout Europe, particularly on the shores of the Baltic and in Northern Italy; in the United States it is rare. The adult worm is from 2 to 9 meters in length, made up of many segments (3,000–4,000). These are commonly broader than they are long. The head is oval, 2–3 mm. in length, without hooklets, and with a lateral groove instead of suckers. The divisions of the uterus are few and simple. The eggs are oval, 45 mm. by 70 mm., of brown color, and in some cases show a small operculum or lid.

(4) Hymenolepsis nana. The rat tape-worm. The smallest tape-worm known for man is but 5 to 45 mm. in length, with from 100 to 200 small segments (see Fig. 101). The head has a rostellum and many

hooklets. The segments show a single, unbranched uterus. The eggs are round, from 50 to 40 micra in diameter, having a double shell, the inner shell showing polar projections with filamentous appendages (see Fig. 102).

The frequency of this parasite has only lately been recognized. It is common in Sicily, 10 per cent. of the children being said to be infected. A number of cases have been reported in the United States.

In the rat the eggs of this worm develop in the villi of the intestine, and the larvæ from this seat easily reach the intestine. Man is pre-
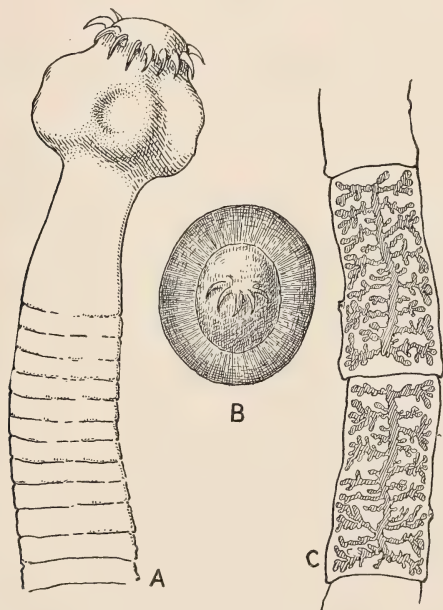
Fig. 100.—Tænia solium. A, head, with hooklets and sucking disks, × 10; B, ovum, × 700; C, proglottides, × .25.

sumably infected through eating food infected with ova from the feces of rats, and a like cycle must be assumed.

Habitat.—The teniæ regularly lodge in the upper part of the small intestine.

Symptoms.—In the great majority of cases tape-worm infection produces no definite symptoms, until segments of the worm are passed and observed in the feces. In nervous women great distress may be produced by the knowledge of this evidence of infection. In other cases vague digestive disturbances are complained of. The excessive appetite commonly believed to be excited by tape-worm infection is wholly legendary. Since microscopic examination of the feces is now generally practiced, the ova of tape-worms are often found unexpectedly.

Severe anemia may be caused by tape-worm infection, particularly by the dibothriocephalus latus. For this reason the feces of all anemic patients should be examined for ova. An eosinophilia is regularly present in the blood.

Diagnosis.—The appearance of segments or ova in the feces is the only reliable evidence. The different varieties of tape-worm are distinguished by details of the structure of the ova, segments, or heads of the worms. Eosinophilia is always suggestive.

FIG. 101.—Hymenolepis nana, enlarged. (International Clinics, Vol. IV, Series 19, page 255.)

Prophylaxis.—The segments of tape-worms should be burned. Infected beef or pork should be rejected in the abattoirs. The cysticerci, if present in numbers, show plainly, especially in the muscles of the tongue and neck. All meat or fish should be thoroughly cooked.

Treatment.—The intestine is emptied by a limited diet for two days or more and the use of mild purgatives. Fasting for 24 hours is sometimes enjoined. Then one of the anthelmintics is given in full dose and followed in 2 or 3 hours by a full dose of castor oil. Of the many anthel-

FIG. 102.—Ova of Hymenolepis nana. (International Clinics, Vol. IV, Series 19, page 260.)

mintics the following are preferred: Extract of male-fern in doses of 1 to 2 drams. Kousso, 5 to 7 drams in an infusion or tablets or capsules. Kamala, 1 to 2 drams in syrup or cinnamon water. Pumpkin seeds, 3 or 4 ounces macerated for 12 hours, and the resulting liquor given.

The worm should be passed into warm water, as contact with cold leads to violent contraction with possible breaking of the chain. The segments should be collected and washed and the head sought. Often it escapes detection, even when passed. Two or three months must then elapse before cure is assured by non-reappearance of segments. If segments reappear, treatment must be repeated.

VISCERAL INFECTIONS

As already pointed out, if the ova of certain tape-worms are taken into the human stomach, the embryos there set free pass through the intestinal wall, make their way through the lymph-channels to the circulation and are distributed to all parts of the body. Lodging in the subcutaneous tissues, muscles or the viscera they form the cyst-like structures characteristic of the larval stage. These cysts may grow to some size and present symptoms as tumors. This chain of events takes place in the case of two known tape-worms, the tenia solium and the tenia echinococcus.

CYSTICERCUS CELLULOSÆ

The ova of tenia solium may reach the human stomach by ingestion, the patient himself or some one in close contact with him harboring the mature worm and passing the ova in the feces, or mature segments containing ova may wander from the intestine into the stomach or may be drawn into the stomach by prolonged vomiting. Cystic tumors containing the embryos may then form in any part of the body. The most frequent seats of lodgment are the subcutaneous tissue, the muscles, the brain or the eye. In the subcutaneous tissue and muscles they form multiple small, movable tumors. If few, no symptoms are excited. If many, there may be pain and stiffness.

The small cysts are most often casually discovered in the course of post-mortem examination. The diagnosis has occasionally been made during life by the excision of one of the nodules and its microscopic examination. In the brain the cysticercus, if lodged in the motor area, may give rise to irritation and produce attacks resembling Jacksonian epilepsy, or general convulsions. In the ventricles they may grow to considerable size. Lodged in the floor of the fourth ventricle the cysts may cause diabetes, persistent vomiting, or respiratory paralysis. The diagnosis is practically never made during life. Before careful inspection of all meats was practiced in Germany, Virchow is said to have found cysticercus in the brain of one out of every 30 post-mortem subjects; after the establishment of inspection, the number fell to one in 280. Brain cysticerci are very rarely observed in this country even in post-mortem work.

In the eye the cysticercus forms a tumor which may be recognized.

ECHINOCOCCUS DISEASE. HYDATID CYST

Tenia Echinococcus.—The mature parasite, a small tape-worm, is normally found in the intestine of the dog, fox, wolf or jackal. The dog is naturally the agent in human infection. In Iceland 28 per cent. of the dogs are infected, in Australia, 40 to 50 per cent., in America but very few. The worm is one of the smallest cestodes, being from 3 to 6 mm. in length. The minute head bears four suckers, a rostellum and a double row of from 28 to 50 hooklets. The body consists of only 3 or 4 segments, which, however, contain great numbers of eggs. The ova

are from 30 to 36 micra in diameter and have a thin striated shell. Through contaminated drinking-water or vegetables, or by direct contact with an infected dog, the ova may reach the human stomach. The shell is then dissolved, the embryo set free, and making its way through the intestinal wall passes into the portal vein and so reaches the liver, or reaching the general circulation through the lymph-channels it may be carried to any part of the body and lodge in the lungs, brain, spleen or any of the other viscera.

The importance of the echinococcus infection rests upon the remarkable evolution which the embryo undergoes when lodged in a favorable site, such as the liver. One month after infection the parasite is represented by a minute cyst, perhaps 1 mm. in diameter, surrounded by a fibrous sheath. Gradually this cyst enlarges and its wall becomes differentiated into two distinct layers, an outer laminated fibrous sheath derived from the connective tissues of the host and an inner germinal layer representing the parasite. The cavity contains clear fluid. As growth continues the cyst continually enlarges, its fibrous sheath thickens and from the germinal layer new cysts are budded off, forming the so-called daughter cysts, each of which is filled with fluid in which float a number of scolices (the head-segments of the adult worm) armed with their characteristic hooklets. The number of daughter cysts produced is apparently without limit. In course of time these daughter cysts become free in the fluid of the primary cyst, and may by their friction upon one another give rise to the peculiar fremitus sometimes detected in hydatid cysts. Also some of the daughter cysts may degenerate and the scolices and hooklets be set free in the fluid of the primary cyst. In this manner a hydatid cyst may grow to form an enormous cystic tumor surrounded by a dense fibrous capsule, the inner part of which is laminated in a manner which renders it pathognomonic of the disease. The fluid of hyatid cysts is clear or slightly yellow in color, neutral or faintly acid, of a specific gravity 1010 to 1015, containing traces of sodium chloride, glucose, inosite, leucin, cholesterin and albumin. The fluid also regularly shows under the microscope the characteristic scolices and hooklets, and crystals of cholesterin. The scolices are small oval or spherical bodies 0.2 to 0.3 mm. in diameter. The rostellum and double row of hooklets may be protruded so as to be readily visible or may be retracted so as to be seen only when one looks directly into the rostellar opening. The presence of scolices and hooklets is diagnostic.

The evolution thus described gives rise to one great cyst containing many smaller ones, the so-called endogenous development. In rare instances some of the secondary cysts penetrate the primary capsule and undergo evolution outside of it, giving rise to an exogenous development, the so-called multilocular echinococcus.

An echinococcus cyst may die, its contents become inspissated into a pasty mass or calcified. If secondary infection with pyogenic organisms takes place at any time, suppuration develops and the cyst becomes an abscess. In rare instances the cyst ruptures and discharges its contents

into the bronchi, the gall ducts, the intestine, the urinary passages, or even the inferior vena cava.

Symptoms.—An echinococcus cyst is a slow-growing tumor. In the liver it causes discomfort or actual pain, indefinite gastric disturbances, and possibly jaundice. It presents itself as an enlargement of the liver, steadily progressive. A tumor may be outlined, and if superficial the peculiar fremitus caused by impact of the daughter cysts upon one another may be felt. The tumor is tense and fluctuation can rarely be determined.

Diagnosis.—Hydatid cysts are most often recognized at operation. If the fluid be aspirated or discharged, the finding of hooklets or scolices is pathognomonic. Even apart from these, the composition is suggestive, the crystals of cholesterin and the presence of sugar being important. Where only remnants of the cyst are obtained, the laminated chitinous membrane is characteristic.

Hydatid cysts of the spleen or peritoneum will present similar symptoms and signs. In the lung the tumors can be recognized only by the discharge of hooklets or scolices in the sputum. The blood may show leukocytosis, and an eosinophilia is regularly present. A complement-fixation analogous to the Wassermann reaction has been demonstrated, employing echinococcus fluid as antigen.

Prophylaxis.—The dog, being the host of the adult worm, should be avoided, particularly in districts where infection is common. The dogs obtain their infection from eating the flesh of sheep, cattle, or hogs suffering from hydatid diseases. Dogs should not be allowed to feed on the refuse of abattoirs.

Treatment.—The cysts must be opened and drained, and the lining membrane removed. If suppuration has taken place, the cyst must be treated as an abscess.

X

DISEASES OF THE NERVOUS SYSTEM

DISEASES OF THE NERVES

NEURITIS

Definition.—An inflammation of a nerve. Two forms are recognized, local, involving a single nerve, and general or multiple neuritis, in which many nerves are affected.

Etiology.—The local form is due (1) to traumatism, such as blows, or lacerations, the pressure of instruments or tumors, or (2) to exposure to cold and wet, or (3) extension of inflammation from adjacent parts, as the facial nerve is involved in caries of the temporal bone. Multiple neuritis may be caused (1) by the toxins of acute infectious diseases, especially typhoid fever or diphtheria, (2) by poisons such as lead, arsenic, mercury, phosphorus, alcohol, or illuminating gas, (3) by cachectic states, such as occur in cancer or tuberculosis, (4) by exposure to cold and wet. The cause of the endemic neuritis known as beri-beri is as yet unknown.

Morbid Anatomy.—The lesions are sometimes interstitial, especially in the localized form, or parenchymatous in multiple neuritis, or both. In the interstitial type the connective tissue between the nerve fibrils is swollen, edematous, and possibly infiltrated with small round cells. The nerve-fibers show some signs of degeneration. In the parenchymatous type the nerve fibrils undergo fatty degeneration and are broken up into globules which stain black with osmic acid. Both the axis-cylinders and their myelin sheaths are destroyed. Later the fatty droplets are absorbed and only the connective-tissue framework of the nerve remains. Some of the changes of the interstitial type are always found in this.

Symptoms.—Localized Neuritis.—Sensory disturbances usually constitute the chief feature, pain in the territory of the nerve, deep and boring in character, tingling and numbness. The nerve itself is tender and may rarely be felt as a thickened cord. Tactile sense may be normal, numbed or lost. Motor disturbances are shown in weakness or loss of power in the tributary muscles. Atrophy follows. Twitchings or contractures of the affected muscles are not uncommon. Trophic changes may appear, the skin becoming shiny, edema appearing and the nails becoming ridged and brittle. The reaction of degeneration may develop in the paralyzed muscles, or the response to the current may be normal. Such localized neuritis is best shown after dislocation of the shoulder, or injuries of the ulna, at the elbow. The duration of symptoms varies from a few days to months.

MULTIPLE NEURITIS.—*Acute Primary Type.*—The disease is ushered in by fever, headache, pains in the limbs and back, all the symptoms of an acute infection. Sensory symptoms (except pains in the limbs) are lacking. Loss of power is noted in the legs and gradually extends upwards, as in Landry's paralysis. The arms, trunk, and even face may be involved. Foot-drop or wrist-drop and loss of reflexes develop.

The disease may be fatal in a few days or the patients recover after weeks or months. Recovery may not be complete.

The disease is with difficulty distinguished from Landry's paralysis or acute anterior poliomyelitis. A definite etiological factor is important. The presence of sensory disturbances and tenderness of the nerves are important. The reaction of degeneration may be present.

Subacute Secondary Type.—This condition follows poisoning by lead, arsenic, or alcohol and the like. Either the sensory or the motor symptoms of a neuritis may be most prominent, although both are present. In lead poisoning the loss of power is most striking; in arsenical or alcoholic poisoning the sensory symptoms are marked. The symptoms of a localized neuritis are present, but not limited to one nerve. The affection is bilateral and symmetrical, both legs to the knees or the arms to the elbows being involved. Rarely are the thighs or shoulders affected. Pain, tingling, numbness and tenderness over the main nerve-trunks, especially in the calves of the legs, are prominent in alcoholic or arsenical cases. In lead poisoning, on the other hand, paralysis is marked often without sensory symptoms. Lead palsy most often affects the wrist, producing wrist-drop, *i.e.*, weakness or disability in extending the wrist, when the hands are held prone with arms stretched forward. Alcoholic or arsenical neuritis, on the other hand, commonly involves the leg, producing foot-drop and the steppage gait. The flexors of the ankle being weak the toes drop when attempt is made to lift the foot as in walking. To meet the difficulty, the knee is raised higher than usual and the foot is thrown outward and forward in a half circle, coming down on the pavement with a flap like a flail. The tendon reflexes (patellar or wrist) are diminished or lost.

Trophic symptoms are often present. Bed-sores may develop. Atrophy of muscles is marked. The reaction of degeneration is regularly present. In the alcoholic cases persistent delirium with hallucinations of sight or hearing may appear. A low fever is occasionally seen, and the patients become greatly emaciated and debilitated. Stupor with loss of control of the sphincters or even convulsions may develop. The condition is most often seen in women who drink moderately but steadily.

The course of the disease varies greatly, but is regularly favorable. After weeks or months the symptoms gradually subside and power returns. Death is possible in very severe cases, especially in the alcoholic type.

The neurons are regenerated from their centers (ganglion cells) in the anterior horns of the spinal cord or on the posterior roots.

The reaction of degeneration is present in the affected muscles.

Faradic excitability is impaired or lost. A stronger galvanic current than usual is required to cause contraction and the action of the poles is reversed so that A C C > K C C, *i.e.*, the anode closure contraction is greater than the cathode closure contraction.

Diagnosis.—The symptoms and local signs of multiple neuritis are characteristic. The reaction of degeneration is important. In some cases without sensory symptoms and little tenderness of the nerves it may be difficult to decide whether the nerve-trunks or the anterior horns of the spinal cord are affected.

From locomotor ataxia careful examination shows marked differences. There is weakness, but not ataxia. The steppage gait is very different from the gait of tabes. Lightning pains are absent. Romberg's sign and pupillary changes are missing.

In alcoholic cases the delirium often causes the neuritis to be overlooked. The combination of wrist- or foot-drop and tenderness of the peripheral nerves with delirium and hallucinations is characteristic.

Treatment.—Rest in bed and careful nursing are most important. In severe cases an air or water bed is desirable. Alcohol, if the cause, should be gradually withdrawn. Local hot applications may relieve the pain. A lotion of menthol and chloral ℥ii of each, in alcohol and camphor water (each ℥i), may be used for the same purpose.

After the acute stage, gentle rubbing, massage, and electricity are indicated. Strychnine is given in full doses.

NEUROMATA

Tumors of nerve-fibers may be composed of nerve tissue, even including ganglion cells, the true neuromata, or may consist of fibrous tissue, the false neuromata.

TRUE NEUROMATA are exceedingly rare, but occur in the thoracic or abdominal cavities on branches of the sympathetic system.

FALSE NEUROMATA are common. They occur early in life and are often congenital. They may be found on any of the cranial or spinal nerves, and vary in size from minute dots to tumors several inches in diameter.

Histologically, they consist of fibrous tissue, derived either from the epineurium, in which case they may hardly involve the nerve-fibers, or the endoneurium, when the nerve-fibers are scattered through or stretched over the tumor.

The growths may be single or occur in thousands.

Solitary Neuromata.—Pain, paresthesia, and muscular spasm in the territory of the affected nerve may be caused by a single neuroma. Loss of sensation or power is rare. Pressure upon the tumor excites or intensifies the pain, while pressure on the nerve-trunk proximal to the tumor relieves it. Neuromata are movable with the nerve from side to side, but not in its length. Excision may be required.

Multiple neuromata occur under several different conditions. (a) Plexiform neuromata, in which multiple tumors occur on all the branches

of a nerve, are most often seen on the head or neck. (b) Tubercula dolorosa, in which multiple tender and painful tumors are found on the terminal branches of cutaneous nerves. (c) General neurofibromatosis or von Reichlinghausen's disease—in which numerous sessile or pedunculated tumors of the nerves are found scattered all over the body. Nevi and skin pigmentation are often associated. Nervous symptoms are rare. The location of the nodules and their histological examination establish the diagnosis.

DISEASES OF THE CEREBRAL NERVES

Disturbances of the cerebral or cranial nerves may be either functional or organic. The functional disturbances are seen particularly in neurasthenia or hysteria and are evidenced by intensified, diminished or perverted function without organic lesion. Organic disturbances are dependent upon lesions either in the nuclei of origin of the nerves, in the course of the nerves through the cranial cavity or its walls, or in their peripheral distribution. The lesions are varied and the disturbances in question are considered in various diseases, such as tumors of the brain, affections of the cerebral vessels, meningitis, fractures of the cranial bones, otitis media, and the like. Certain features of the affections of the cerebral nerves can, however, be best treated by considering them separately and individually.

THE OLFACTORY NERVE AND TRACT

The olfactory centers of the brain are localized in the hippocampal gyrus and the adjoining convolutions. From these the olfactory tracts pass forward on the under surface of the frontal lobe to the olfactory bulb, which lies on the cribriform plate of the ethmoid bone. From the bulb a series of twenty or more fine nerve-bundles pass through the cribriform plate and are distributed to the upper part of the nasal septum and to the outer walls of the nasal fossa as far down as the lower border of the superior turbinate bone. The remaining portions of the nasal mucous membrane are supplied with sensory branches from the fifth nerve, but at present no part in olfactory sensation is attributed to them.

ANOSMIA, loss of the sense of smell, may be congenital, from lack of development of the olfactory nerves, or acquired. The acquired form may be purely functional, as in hysteria or neurasthenia, or organic. The organic lesions most frequently causing anosmia are diseases of the nasal mucous membrane, such as acute or chronic rhinitis, or diseases of the nasal bones, fractures of the base of the cranium involving the anterior fossa, or pressure upon the olfactory bulb or tracts by hemorrhagic or inflammatory exudates or tumors of the brain. Temporary loss of the sense of smell is common after attacks of influenza. Paralysis of the fifth nerve, causing cessation of nasal secretions, may indirectly cause anosmia.

HYPEROSMIA, abnormal acuteness of the sense of smell, is usually **functional.**

PAROSMIA, a perverted sense of smell or olfactory hallucination, **is** rare, but may arise from irritation of the olfactory nerves caused by **the** pressure of tumors, or may occur as an epileptic aura, and has been observed in locomotor ataxia.

The treatment of these conditions must be that of the underlying cause.

OPTIC NERVE AND TRACT

The primary neurons of the optic tract lie, for the most part, in the retina. Thence the nerve-fibers pass backward to the chiasm, where the internal fibers pass to the other eye, the external to the optic tract of the same side, while the middle fibers cross into the opposite tract and are distributed to the external geniculate body, the pulvinar **of**
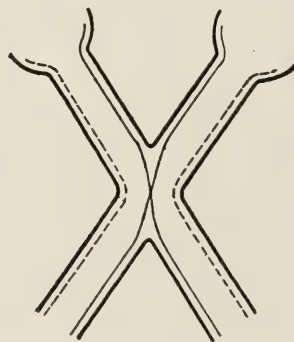
FIG. 103.—The paths of the nerve-fibers in the optic chiasm. The fibers from the outer **halves** of the retinæ do not cross and lie in the corresponding outer portion of the chiasm; the fibers **from the** inner halves decussate in the central part of the chiasm.

the optic thalamus and the superior corpus quadrigeminum. These centers are in turn connected by a secondary neurone system with the cortex of the cuneus and adjacent regions. (See Fig. 103.)

Examination of the eyes, particularly of the retina, is of the utmost value in many cases of systemic disease.

Retinitis.—Notable changes in the retina are common in Bright's disease, syphilis, leukemia and anemia. These changes include (1) cloudiness of the retina, as a whole, due to serous exudation, (2) blurring of the disk for the same reason, (3) engorgement of the veins, and possibly (4) hemorrhages. These hemorrhages show as dots or larger areas, red or almost black in color, depending upon their age. With them are frequently associated (5) white patches due to degeneration or atrophy of the retinal elements caused by previous hemorrhages.

Several types of retinitis are recognizable. (a) Albuminuric. **In** from 15 to 25% of cases of chronic nephritis, especially of the interstitial type, retinal changes are present. Vision fails as the retinitis advances. The retinal cloudiness, hemorrhages and white spots **are**

usually all present. The changes in the disk are commonly less marked, but they may be extreme, and the condition may then hardly be distinguishable from ''choked disk'' due to increased intracranial pressure.

Retinitis in Bright's disease is always of grave import. Recovery may follow, but if the changes are marked death ordinarily follows within six months or a year.

(b) Syphilitic. Retinitis is common in both hereditary and acquired syphilis. Chorioiditis and often iritis are associated with it. It is marked by congestion and haziness of the disk, scattered gray or white spots, often fringed with pigment and circumscribed exudations, along the lines of the larger blood-vessels, forming white lines.

(c) Diabetic. This may be the same as the albuminuric type, or it may be marked by small, bright, white spots above the macula and punctate hemorrhages, but without swelling or haziness of optic nerve or retina.

(d) Leukemic. Marked swelling of the disk and retina and many hemorrhages are present. The vessels are large and tortuous, the blood pale. The fundus is pale and white or yellow spots of exudation, foci of leukocytic infiltration, are seen.

Papillitis (choked disk).—The disk is swollen, prominent, white or gray in color, and often presents white spots and hemorrhages. The retinal veins are distended and tortuous, and both veins and arteries appear broken or interrupted in their course. The retina is swollen, hazy, and presents white patches and hemorrhages. Increased intracranial pressure is the usual explanation, especially that produced by tumor of the brain, but in some cases no adequate cause can be found.

ATROPHY of the optic nerve is either primary or secondary to papillitis. The optic disk appears paler than normal, the color varying from gray to greenish-white. The center is sunken and the lamina cribrosa may appear. The arteries appear narrow. Primary atrophy occurs in tabes, or paralytic dementia, and rarely in multiple sclerosis. Secondary atrophy follows optic neuritis (papillitis), embolism of the central artery, glaucoma and retinitis pigmentosa.

AMBLYOPIA and AMAUROSIS without retinal changes occur in a variety of conditions, uremia, diabetes, malaria, anemia, poisoning by methyl-alcohol, quinine and the like. Hysteria or neurasthenia may cause these conditions.

Hemianopsia.—A lesion of either optic nerve anterior to the chiasm will produce blindness in the corresponding eye. A lesion of the chiasm itself so situated as to involve the fibers passing to the nasal half of each eye will cause insensitiveness of those parts, with the result that the outer or temporal half of the visual field is not appreciated. This condition is called bitemporal hemianopsia.

A lesion of either optic tract behind the chiasm produces loss of the same half of the visual field in both eyes, or bilateral homonymous hemianopsia.

Hemianopsia is therefore of importance in the localization of intracranial lesions. It may, however, be produced by functional disturbances, such as megrim, hysteria or neurasthenia.

THE OCULOMOTOR, TROCHLEAR AND ABDUCENS NERVES

(Third, Fourth and Sixth Nerves)

The nuclei of origin of the third and fourth nerves lie in the floor of the aqueduct of Sylvius, of the sixth in the floor of the fourth ventricle. The third emerges on the inner aspect of the crus just behind the optic chiasm. The fourth winds around the crus and appears on its outer side; the sixth emerges from the groove between pons and medulla. All three pass forward in the outer wall of the cavernous sinus, through the sphenoidal fissure, into the orbit, where they are distributed to the several muscles of the eye. The third supplies all the muscles except the external rectus, supplied by the sixth, and the superior oblique, supplied by the fourth. These nerves may either partially or completely lose their function, and produce corresponding paresis or paralysis of the dependent muscles. The nerves may be affected by lesions of their centers or of any part of their trunks. Tumors of the brain, lesions of the blood-vessels, meningitis, or peripheral neuritis, as in diphtheria, are most frequently encountered. The possibility of congenital strabismus or inequality of the pupils must always be borne in mind.

Paralysis of one or more ocular muscles produces (1) an abnormal position of the globe of the eye or dilatation of the pupil from contraction of the unopposed antagonists. (2) Impairment or loss of certain motions of the eye. (3) Diplopia. (4) Secondary deviation. If, with the sound eye covered, the patient fixes the affected eye upon an object before him, the sound eye will be found displaced in the direction of the action of the weak or paralyzed muscle. Thus, if the right internal rectus is affected, the left eye will, under these conditions, be found displaced outward. (5) Erroneous projection, and possibly vertigo.

Paralysis of the third nerve results in ptosis, the eyeball is rotated outward and almost immovable, motion of the globe upward, downward or inward being particularly weak. Dilatation of the pupil may or may not be present.

Paralysis of the fourth nerve alone causes but slight disturbance of motion. On looking downward the eye turns slightly inward and diplopia results. Thus on going downstairs the steps appear double.

Paralysis of the sixth nerve produces internal strabismus, with inability to rotate the eye outward. Paresis of ocular nerves results in varying degrees of the disturbances caused by complete loss of function.

Pupillary Irregularities.—Inequality of the pupils (anisocoria) of moderate degree is common in healthy persons. Marked inequality

usually indicates some definite lesion. It is most often associated with meningitis or cerebral hemorrhages, tabes, dementia paralytica, or tumor of the neck or upper part of the cord.

The ARGYLL-ROBERTSON PUPIL is indicated by a loss of the normal response to light with preservation of pupillary contraction in accommodation. Myosis is usually associated. The condition is closely associated with syphilis, and is most frequent in tabes dorsalis.

NYSTAGMUS, rhythmical oscillation of the eyes, usually from side to side, is met with in a variety of conditions. It is common in the blind or those with defective vision. It is most frequent in multiple sclerosis or lesions in the posterior fossa, especially those involving the cerebellum.

The treatment of these conditions depends upon the cause. Persistent ocular palsies demand correction by prisms.

TRIGEMINAL NERVE

(Fifth Nerve)

This nerve consists of both sensory and motor elements. The sensory portion supplies the orbit, the face, the scalp from the vertex to the ramus of the inferior maxilla and the mucous membranes of the nose and mouth. From the Gasserian ganglion these fibers pass to the pons, where they end in an extensive center. The motor fibers also find their center in the pons. Of the three branches the ophthalmic leaves the cranial cavity through the sphenoidal fissure, the superior maxillary through the foramen rotundum, the inferior maxillary (the motor portion) through the foramen ovale.

This nerve may, therefore, be involved in lesions of the pons, by tumors or meningitis at the base of the brain, or fractures of the base (very rarely). The Gasserian ganglion is the probable seat of the lesions underlying herpes labialis and facial neuralgia.

PARALYSIS of the fifth nerve shows itself by loss of sensation in the face and anterior portion of the scalp, by some impairment of smell due to dryness of the nasal fossæ, and taste may be impaired over the anterior two-thirds of the tongue. The muscles of mastication are paralyzed, as can be appreciated, by placing the fingers just below the inferior maxillary articulation during the action of chewing. The lower jaw can be moved only to the sound side, and the mouth cannot be widely opened (digastric and mylohyoid paralysis).

TRISMUS, tonic spasm of the muscles of the lower jaw, is a symptom of tetanus and sometimes of tetany. It may also occur in pontine lesions, as a reflex from carious teeth or periostitis of the jaw, or in hysteria. Clonic spasms, with rhythmic movements of the jaw, occur in meningitis, convulsions, epilepsy, hysteria and paralysis agitans. "Grinding the teeth" during sleep is a common occurrence in children, restless or disturbed for any reason.

THE FACIAL NERVE

(Seventh Nerve)

The facial nerve nucleus, after the manner of the cerebral nerves, has a center in the floor of the fourth ventricle (pons Varolii), but it has also a definite cortical center in the lower portion of the motor area ventral to the fissure of Rolando. Axones passing from cells in this area follow the motor tract to the pons, crossing just before reaching the nucleus. Emerging from the lateral aspect of the pons the nerve passes into the Fallopian canal, around the inner wall of the tympanum and emerges from the stylomastoid foramen. The nerve supplies the muscles of expression, the platysma, part of the digastric and stylohyoid muscles, and gives a small twig to the stapedius muscle of the ear.

Paralysis of the facial (Bell's palsy) nerve may result from (1) lesions of the cortex or motor tract above the pons (supranuclear paralysis). (2) Lesions of the nucleus (nuclear paralysis). (3) Lesions of the nerve-trunk (infranuclear or peripheral paralysis).

1. SUPRANUCLEAR PARALYSIS is regularly part of a hemiplegia, and may be caused by any of the lesions producing that condition, hemorrhage, tumors, etc. The facial palsy is on the same side as the paralysis of arm and leg. It is distinguished from peripheral palsy by the preservation of the electrical reactions, and the fact that, as a rule, the orbicularis palpebrarum, frontalis and corrugator muscles are spared.

2. NUCLEAR PARALYSIS, due to lesions in the pons, will regularly be accompanied by affections of the fifth, sixth or eighth nerves from involvement of the adjacent nuclei. Otherwise it does not differ from peripheral palsy. Crossed paralysis, paralysis of the seventh nerve on one side with loss of power in arm and leg of the other side, results from lesions in the lower half of the pons such that they involve the nucleus of the seventh nerve and the motor tract, the fibers of which have not yet crossed, the decussation occurring in the medulla.

3. PERIPHERAL PARALYSIS. (1) The nerve may be involved in processes at the base of the brain, meningitis, tumors, or fracture.

(2) In the Fallopian aqueduct, inflammatory processes (otitis media, or caries of the bone) may involve the seventh nerve. The eighth nerve may suffer at the same time, with resulting deafness.

(3) In the neck paralysis may result (a) from trauma of any kind or division during operations; (b) from syphilis, especially in the secondary period; (c) from exposure to cold; (d) it may be associated with herpes.

Symptoms.—Facial paralysis is usually diagnosticated on sight. The affected side of the face appears flat, and the lips are drawn toward the sound side. If the paralysis involves all branches, the brow cannot be wrinkled, frowning is impossible, the eye is imperfectly closed, sniffing, whistling and speaking are impaired. In supranuclear paralysis, the upper branches often escape, and the forehead and eye may not be affected.

Certain points may closely localize the site of the affection. If the nerve is involved between the genu and the origin of the chorda tympani, the sense of taste may be lost on the anterior two-thirds of the tongue. Lesions beyond that point have no such influence.

If the lesion is located centrally to the origin of the nerve supplying the stapedius muscle, increased sensitiveness to low tones may be noted. In the supranuclear type the electrical reactions remain normal; in the nuclear or peripheral palsy the reaction of degeneration obtains.

Course.—Facial palsy regularly recovers. In hemiplegia the recovery may be only partial, but is usually rapid. Traumatic palsy may, however, be permanent.

Treatment.—In most cases no treatment is required. The underlying cause, such as syphilis or otitis media, should be appropriately treated. Electricity and massage may be employed as for other paralyses.

Spasm.—Localized or general spasm of the facial muscles may occur. These usually belong in the category of habit spasms ordinarily affecting the muscles of the eye and adjacent parts of the face (blepharospasm). There is rapid, rhythmic action of the affected muscles.

Treatment.—Any source of irritation, especially rhinitis or adenoids, must be removed. The general condition of the patient must be considered and measures employed to correct nervousness. In severe cases division of the nerve may be required.

THE AUDITORY NERVE
(Eighth Nerve)

The spinal centers of the eighth nerve are in the lower portion of the floor of the fourth ventricle. The nerve emerges from the medulla just below the pons Varolii, passes forward, enters the auditory canal, and at its bottom divides into two branches: the cochlear, concerned with hearing, and the vestibular, whose function is the maintenance of equilibrium.

The cochlear branch is more frequently affected in its course than the vestibular. The cortical center for hearing is the superior temporal convolution. The cells of this center are connected by their axones with the spinal centers. Lesion of the center or of this tract produces the condition of word-deafness in which words are heard as sounds, but not understood.

Either branch of the nerve may be involved in the degenerations complicating cerebrospinal fever and diphtheria, the cochlear especially, with resulting deafness. Tumors, hemorrhage, or fracture of the base of the cranium may involve the whole nerve. A primary degeneration sometimes occurs in locomotor ataxia.

Hyperesthesia of the eighth nerve, or hyperacusis, is common in headache, in hysteria, or neurasthenia. It rarely results from cerebral disease. Tinnitus aurium includes various subjective sensations of ringing, roaring, ticking or buzzing noises in the ear. The disorder

most frequently results from neurasthenia, but may be caused by the pressure of wax or by definite disease of the middle or internal ear.

Deafness may result from various causes. It is important to test the integrity of the nerve. This may be done by employing a tuning fork or watch, comparing the hearing under normal conditions with that obtained by closing the external meatus and holding the fork or watch against the mastoid process. If the cochlear nerve itself is diseased, the sound is not heard under the latter circumstances. Under normal conditions the sound is readily transmitted through the bone to the cochlea. If bone transmission is better than air there is then some obstruction or disease of the middle ear.

The vestibular nerve is rarely affected. Disease of the vestibular portion gives rise to vertigo, nystagmus and loss of co-ordination of the muscles of the head, neck and eyes.

Vertigo is dependent upon some disturbance of the complex muscular adjustments by which equilibrium is maintained in various positions. There are many causes of vertigo. It is a common symptom of neurasthenia and hysteria, of muscular and other eye defects, of disease of the internal or middle ear, of tumors of the brain (especially those involving the cerebellum), and other cerebral lesions, such as arteriosclerosis. It may result from cardiac disease or gastro-intestinal disorders, particularly autointoxication.

Ménière's Disease.—An affection described by a French physician, characterized by attacks of vertigo and tinnitus, associated with vomiting and sometimes loss of consciousness. The paroxysms occur irregularly, sometimes several in a day, sometimes at long intervals. The affection has been attributed to disease of the labyrinth, but the pathology is unsettled. The more carefully vertigo is studied the more frequently definite cause is found, and the diagnosis of Ménière's disease is now rarely heard.

Treatment.—Careful examination for local disease of the ear and appropriate treatment of any lesion there present are essential in both tinnitus and vertigo. If further examination shows other definite conditions, such as neurasthenia, arteriosclerosis, disease of the heart which may be causative, treatment must be planned accordingly.

For Ménière's disease various remedies are recommended, such as bromide or iodide of potassium; the salicylates and quinine have been employed with doubtful efficacy.

THE GLOSSO-PHARYNGEAL NERVE

(Ninth Nerve)

The center for the ninth nerve lies beneath the fovea in the floor of the fourth ventricle. It leaves the cranium by the jugular foramen, and supplies motor and sensory branches to the pharynx and adjacent parts. Its most disputed function lies in supplying the sense of taste to the posterior one-third of the tongue.

Symptoms.—The pharyngeal symptoms of bulbar paralysis are explained by lesion of the ninth nerve. The nerve may be affected by tumors or meningitis, causing difficulty in *swallowing.*

Disturbances of taste on the posterior part of the tongue result from lesions of the ninth nerve. These are tested for by the application of solutions of quinine (bitter), saccharin (sweet), vinegar (acid) on the protruded tongue. The tongue must be held out till decision is given. A feeble galvanic current giving rise to a metallic taste may be employed. *Salivary secretion* may be affected by disease of this nerve.

THE PNEUMOGASTRIC NERVE

(Tenth Nerve)

The center of the vagus lies in the floor of the fourth ventricle, so closely related to that of the ninth as to be with difficulty distinguishable from it. The nerve leaves the cranium through the jugular foramen, and has the widest distribution of any of the cranial nerves. It contains both motor and sensory fibers, giving motor supply to some of the pharyngeal muscles, to the larynx, trachea and bronchi, and to the whole gastro-intestinal tract excepting the rectum, and sensation to the same general territory, and also the dura, ear and pericardium. The cardiac nerves also form part of the vagus.

Symptoms.—The nerve may be involved at its center, especially in the glosso-labio-laryngeal paralysis, in diphtheria, anterior poliomyelitis and the like. At the base of the brain it may be compressed by tumors, or involved in meningitis.

In the neck it is subject to traumatism, blows or gunshot wounds, the pressure of tumors or section in the course of operations, or it may be affected by neuritis.

The symptoms will depend upon the particular branches affected.

PHARYNGEAL BRANCHES.—Paralysis follows diphtheria, meningitis, tumors, bulbar palsy and the like. Difficulty in swallowing results. Food may pass into the larynx or be regurgitated through the nose. Spasm of the pharynx occurs in hysteria (globus hystericus) and other nervous affections, especially hydrophobia.

LARYNGEAL BRANCHES.—*Paralysis of the larynx.* Several types of laryngeal paralysis are recognized.

In general hoarseness, aphonia, cough, stridulous respiration or dyspnea may be caused by laryngeal paralysis.

Laryngoscopic examination is necessary to determine which muscles are affected.

Bilateral Abductor Paralysis.—The cords are approximated and cannot be abducted. Stridulous respiration and severe dyspnea result, while phonation is preserved. The condition develops from involvement of the center in bulbar palsy, or tabes. It may be caused by pressure on both vagi, or by exposure to cold. Some cases are hysterical.

Unilateral Abductor Paralysis.—This results from pressure on one

nerve, especially upon the recurrent laryngeal by aneurisms. The left nerve is most frequently involved. The cord on the affected side does not move in respiration or phonation.

Adductor Paralysis.—The cords cannot be approximated and phonation is impaired or lost. Hysteria is the usual cause.

Spasm of the larynx occurs in laryngismus stridulus, and is undoubtedly present in most cases of acute laryngitis (croup), diphtheritic or not, in children. For this reason the relaxation produced by nausea and vomiting gives at least temporary relief in such conditions. Paroxysmal attacks of laryngeal spasm explain the laryngeal crises of tabes.

Anesthesia of the larynx occurs in bulbar palsy and in diphtheritic neuritis. Dysphagia is also present. Food may enter the respiratory passages and bronchopneumonia result. The condition may be hysterical.

CARDIAC BRANCHES.—*Motor.* Vagus stimulation may cause slowing of the heart action, or paralysis may result in great rapidity, but neither of these conditions is ordinarily realized unless both nerves are involved. For this reason disturbances of the heart definitely traceable to vagus injuries are rare, except as part of a diphtheritic or other general neuritis.

Sensory cardiac disturbances are treated under "Neuroses of the Heart."

PULMONARY BRANCHES.—Bronchial asthma may be due to spasm of the bronchial muscles produced by vagus irritation. Slowing or quickening of respiration may result from disease of these branches, but again only in case of involvement of both vagi.

ESOPHAGEAL AND GASTRIC BRANCHES.—Spasm of the esophagus is attributable to vagus irritation. The various neuroses of the stomach are evidences of disturbance of vagus function. The gastric crises of tabes are doubtless due to central lesions.

THE SPINAL ACCESSORY NERVE

(Eleventh Nerve)

The smaller part of this nerve joins the vagus, and is considered as part of it. The spinal portion supplies the sternomastoid and trapezius muscles, and is purely motor. The center for the spinal accessory is very extensive, extending from the medulla to the fifth or sixth cervical segment. The nerve passes through the jugular foramen, pierces the sterno-mastoid in the neck, and crosses the occipital triangle to end in the trapezius.

Paralysis of one spinal accessory nerve is shown by disability in rotating the head to the other side (sterno-mastoid), by drooping of the shoulder, inward rotation of the angle of the scapula, and disability in raising the arms (trapezius). Bilateral palsy results in loss of power to lift the head (sterno-mastoid) or to prevent it falling forward (trapezius).

Paralysis results from disease of the spinal cord, progressive muscular atrophy (bilateral palsy), cervical myelitis, syringomyelia and pachymeningitis, or caries of cervical vertebræ (tuberculosis). In the latter cases the lesion is usually one-sided.

Torticollis or Wry-neck.—The abnormal position of the head and face caused by unequal action of the cervical muscles is in large part due to the action of sterno-mastoid and trapezius, though other muscles of the neck are involved.

Congenital cases are due to lack of development or injuries of the sterno-mastoid at birth. Facial asymmetry is usually present. It is commonly not noted at birth, and may not be for several years. It is regularly relieved by tenotomy.

Acquired torticollis or temporary wry-neck may be one form of muscular rheumatism or may be caused by adenitis, the nodes underlying the sterno-mastoid being tender and causing spasm of the muscle, or by caries of cervical vertebra.

Spasmodic wry-neck occurs from unknown causes and the lesions are unknown. The condition is chronic. The head is drawn downward and toward the affected side, the chin and face upward and toward the opposite side. Other muscles than those supplied by the spinal accessory are involved. The condition is usually a tonic spasm of the affected muscles, but clonic movements are seen in some cases, and in rare instances the clonic spasms are the more important feature.

Treatment.—The temporary form is to be treated according to the cause, as rheumatic or gouty, or due to adenitis or caries. In acute cases rest is most important. This may be secured merely by lying with the head in a soft pillow, or by sand bags placed at the sides of it. Heat and gentle friction are grateful. Aspirin or salicylates may be given, as for muscular rheumatism.

If adenitis or caries of the vertebræ is present, treatment must be planned accordingly. For the spasmodic form, the only effective treatment is orthopedic or operative.

HYPOGLOSSAL NERVE

(Twelfth Nerve)

The cortical center is the lower part of the anterior central gyrus.

The spinal center is in the floor of the lower portion of the fourth ventricle and the adjacent part of the medulla. The nerve leaves the spinal canal by the anterior condyloid foramen, lies deeply in the neck, and passes forward near the angle of the jaw to supply the muscles of the tongue, its function being purely motor.

Lesions of the cortical center are part of a hemiplegia. Unilateral paralysis without atrophy or reaction of degeneration results.

Nuclear lesions are most common in bulbar paralysis and progressive muscular atrophy. The nerve may be involved by tumors or basal meningitis, by trauma or lead poisoning. The lesions may affect one or both nerves.

In one-sided paralysis of the hypoglossal the tongue when protruded deviates toward the affected side. Atrophy occurs in the peripheral or nuclear lesions. •

In bilateral paralysis the tongue cannot be protruded, is atrophied, and there are difficulties in swallowing and speech. Fibrillary tremors may also be seen.

Spasm of the glossal muscles is rare. It may occur as part of chorea, hysteria, epilepsy or other convulsive disorder; it may be either unilateral or bilateral. A clonic form marked by rapid, rhythmic protrusion of the tongue has been observed. Recovery is the rule in all cases.

DISEASES OF THE SPINAL NERVES
CERVICAL PLEXUS

Neuralgia may affect the nerves of this region, causing pain in the nape of the neck and the occiput. Neurasthenia is the most frequent cause, but caries of the spine, arthritis deformans, tumors and aneurisms of the vertebral artery must be excluded. Tender points midway between the mastoid and the first cervical vertebra may be present. Organic lesions having been excluded, treatment must be that of neuralgia.

Phrenic Nerve.—PARALYSIS of one or both phrenic nerves may occur. Paralysis of the phrenic nerve may result from involvement of the anterior horns at the level of the third or fourth cervical nerves, from anterior poliomyelitis or ascending paralysis or multiple neuritis, such as follows diphtheria or lead poisoning. In rarer cases it follows vertebral caries or pachymeningitis.

Unilateral paralysis gives but few symptoms, and is often overlooked. Bilateral paralysis shows itself by immobility of the diaphragm. Respiration is thoracic, and the liver and spleen are no longer pushed down by inspiration. The epigastrium is depressed in inspiration and rises in expiration. Dyspnea and cyanosis develop in some cases. Coughing is difficult and mucus collects in the bronchi.

THE DIAGNOSIS.—Immobility of the diaphragm from other causes, diaphragmatic pleurisy, emphysema, large pleural effusions must be excluded. Many women use the diaphragm poorly under normal conditions. The development of signs of phrenic paralysis in the course of anterior poliomyelitis, or ascending paralysis, or multiple neuritis is important. Treatment is that of the underlying condition. Thoracic respiration should be favored. In severe cases the head should be lowered or the feet raised to favor the expulsion of mucus from the bronchi.

HICCOUGH.—This is caused by an associated sudden contraction of the diaphragm and closure of the glottis, often repeated rhythmically.

The exciting causes are varied, and may be grouped thus: (a) Irritative, the spasm being excited by some irritation in the territory of the pneumogastric, such as too hot food, local disease of the esophagus

near the diaphragm, gastric or intestinal indigestion, any abdominal inflammation, especially gastritis or peritonitis. (b) Constitutional, arising from gout, diabetes or chronic nephritis. (c) Neurotic, in such conditions as hysteria, epilepsy and brain tumors.

Treatment.—Holding the breath as long as possible will stop mild attacks. Washing out the stomach, an ether spray on the abdomen, the hypodermic use of apomorphine, morphine, or pilocarpine, inhalations of ether, chloroform, or amyl nitrite, galvanization of the phrenic nerve, and pressure upon the nerve between the heads of the sterno-mastoid, all have been tried.

THE BRACHIAL PLEXUS

Tumors, enlarged glands or aneurisms may affect the brachial plexus by pressure. Inflammation is very rare, but may follow an ascending neuritis. Trauma in one or another form is the common cause of partial or complete paralysis. Dislocations of the humerus, especially the subcoracoid, blows on the shoulder, improper crutches, traction in the axilla during delivery, the pressure of a cervical rib, have all caused such disturbances. Loss of power in the arm, partial or complete, is the chief symptom. Sensory symptoms, such as tingling or numbness, may occur. The entire plexus or one of its branches may be involved, the distribution of paralysis and anesthesia varying accordingly. The result of the involvement of the more important nerves may be summarized as follows:

(a) The Long Thoracic. The serratus magnus is paralyzed, and in consequence the angle and posterior border of the scapula stand out from the chest "like wings," especially when the arms are extended forward. The paralysis is usually associated with that of other muscles of the shoulder girdle.

(b) Circumflex Nerve. The deltoid and teres minor are paralyzed. Wasting is marked; the arm cannot be raised; sensation is commonly impaired.

(c) Musculo-spiral Nerve. This nerve is especially easily injured, as it winds around the upper arm, so that paralysis has resulted from sleeping with the arm over the back of a chair or lying on the arm, or allowing the arm to rest on the edge of the table during operations. Lead or alcoholic poisoning is a frequent cause. Wrist-drop and inability to extend the first phalanges are characteristic. Sensory disturbances are rarely marked. The reaction of degeneration develops in severe cases.

(d) Ulnar Nerve. Paralysis may result from neuritis or trauma. The first phalanges cannot be flexed, the others cannot be extended, and claw-hand or "main en griffe" results. The hand is drawn to the radial side and adduction of the thumb is impossible. Sensation may be lost on the backs of two and a half fingers on the ulnar side, and on the palmar surface of one and a half.

(e) Median Nerve. Paralysis results from trauma or neuritis, and

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rarely is the median alone involved. The forearm cannot be pronated more than half way. The wrist flexes toward the ulnar side. The second phalanges of all the fingers and the terminal ones of the index and middle cannot be flexed. The proximal phalanges can be flexed through the interossei. The thumb cannot be flexed or opposed. The wrist can be flexed only when adducted. Sensation is lost or impaired both front and back, except over the little and the inner half of the ring finger. Atrophy is marked.

The prognosis depends upon the cause and the severity of the lesion. Recovery is the rule.

LUMBAR PLEXUS

Branches of this plexus may be affected by the pressure of tumors, lymph-nodes or psoas abscess, or by caries of the vertebræ. The obturator nerve may be injured in parturition and paralysis of the adductors, shown by inability to cross one leg over the other, result. The anterior crural may be impaired by wounds or dislocation of the hip joint. Paralysis of the extensors of the knee with wasting and anesthesia on the inner aspect of the thigh and leg results. The external cutaneous nerve may be affected by neuritis. Tenderness of the nerve as it passes under Poupart's ligament just internal to the anterior superior spine is found, and there are pain and paresthesia in the territory of the nerve, lasting indefinitely. Division of the nerve has been resorted to.

SACRAL PLEXUS

The branches of this plexus may be injured by pelvic tumors or inflammations, or during parturition. Neuritis may follow sciatica.

The great sciatic nerve. The flexors of the leg and all the muscles below the knee are paralyzed, and there is anesthesia of the outer half of the leg and both sole and dorsum of the foot.

The external popliteal nerve. Paralysis is common in peripheral neuritis. The peronei, the tibialis anticus and both extensors of the toes are paralyzed. Drop-foot and steppage gait result. Sensation is lost on the outer half of the front of the leg and the dorsum of the foot.

Internal popliteal nerve. Paralysis is rare. The foot and toes cannot be extended. The patient cannot stand on tip-toe. In severe and old cases talipes calcaneus results.

SCIATICA

A neuritis or neuralgia of the great sciatic nerve. Adult and vigorous men and women suffer from the affection. Exposure to cold, overexertion, gout and rheumatism are regarded as causative factors. Pressure of spinal or pelvic tumors, especially those of the uterus or ovaries, new growths, impacted feces and the like may cause sciatica. In many cases no satisfactory cause is to be found. Anatomically both

a perineuritis and interstitial neuritis have been found in the few cases examined.

Symptoms.—Pain in the course of the nerve, mild or severe, constant or intermittent, is present. Beginning at the upper part of the nerve the pain extends till it affects the whole territory. Sensitive points over the course of the nerve are present. Walking increases the pain, and seeking relief the patient walks on the toes. In severe cases the patients are bed-ridden.

Wasting and cramps occur in prolonged cases. Flexion of the thigh with the leg straight increases the pain.

Duration and course are both variable. Sciatica may last weeks, months, or even years. Relapses are common.

In many cases the affection seems to gradually lessen with little regard to treatment.

Diagnosis.—Disease of the spine must be carefully sought for. Pelvic tumors must be excluded by examination of the abdomen, and rectum or vagina. Hip-joint disease must be excluded by freedom of motion in the joint, so long as the thigh is not flexed. Attempt to flex the thigh with the leg straight increases the tension on the sciatic nerve and elicits pain in sciatica. Sacro-iliac disease must be excluded by the harmlessness of pressure on the sides of the pelvis and the absence of local signs.

Tender points along the course of the sciatic nerve help to confirm the diagnosis.

Treatment.—Rest is the prime factor. Careful dieting, sunshine and fresh air are important. A long splint may be employed. Hot water, poultices of flaxseed or mustard, blisters, afford temporary relief. The thermo-cautery has been much used, but the continuous heat of poultices is more successful. Phenacetin or antifebrin give only temporary relief, and their continued use is depressing. Cocaine or morphine hypodermatically may be employed as last resorts, but the danger of habit is great.

Injections of distilled water, alcohol or even choloroform into the nerve have been practiced.

The galvanic current has sometimes given relief. Nerve stretching by cutting down on the nerve and making sufficient traction upon it to lift the leg has been successful.

In the chronic cases attention to feeding and the beneficent influences of fresh air and sunshine are more important than local measures. In the very thin forced feeding is of value.

DISEASES OF THE SPINAL CORD AND MENINGES

SPINAL PACHYMENINGITIS

Definition.—An inflammation of the dura of the cord, on either its external or internal surface.

Etiology.—External pachymeningitis occurs in conjunction with

spinal injuries and tumors or inflammations (tuberculosis especially) of the vertebræ. It may be acute or chronic, depending on the activity of the bone disease.

Internal pachymeningitis occurs as part of the external processes, but there is also an independent form, analogous to the hemorrhagic pachymeningitis of the brain, which occurs especially in syphilitic, alcoholic or insane subjects.

Morbid Anatomy.—The dura is much thickened and an exudate is found on its external or internal surface or both. The exudate is usually purulent or hemorrhagic. In tubercular cases it may be cheesy. In the hemorrhagic form the internal surface is coated with a delicate vascular membrane, and there are punctate or larger hemorrhages. Blood cysts are sometimes found in old cases.

Symptoms.—These include: (1) Pain, hyperesthesia or anesthesia in the area of the nerves whose roots are pressed upon. The pain may be very severe. (2) Spasm, weakness or paralysis and atrophy in affected muscles. The arms are most often involved and typical "main en griffe" may be produced. (3) Secondary degeneration of the cord may follow, with involvement of the legs, ending in a spastic paraplegia.

Diagnosis.—An X-ray examination of the spine should be made to exclude tubercular disease or tumors. The Wassermann reaction and a spinal lymphocytosis should be present in syphilis.

From amyotrophic lateral sclerosis the disease is distinguished by the marked sensory disturbances. From syringomyelia by the preservation of temperature sense. Tumors are excluded with difficulty, as the symptoms are identical.

Prognosis.—The disease is chronic—lasting several years—and is usually fatal from intercurrent infections, especially of the bladder and urinary tract, or from exhaustion.

Treatment.—If syphilis is present, a thorough anti-syphilitic treatment is indicated. (See page 450.) Otherwise counter-irritation by blister or the actual cautery, rest and symptomatic treatment must be relied on.

SPINAL LEPTOMENINGITIS

Definition.—An acute or chronic inflammation of the pia mater of the cord.

ACUTE SPINAL LEPTOMENINGITIS

Etiology.—The pia mater of the cord being continuous with that of the brain is regularly involved with it. Acute spinal leptomeningitis is therefore usually part of a cerebral meningitis, and is dependent on the same causes. (See page 524.) An acute suppurative inflammation of the spinal meninges may occur independently in the terminal stages of some chronic diseases, such as chronic nephritis, or from the invasion of suppurative processes involving the spinal vertebræ.

Morbid Anatomy.—The lesions are those of the corresponding cerebral process.

Symptoms.—These are part of the general picture of cerebrospinal meningitis. Rigidity of the neck, spasm or paralysis of muscles of the trunk or extremities, the sensory and vasomotor disturbances of these parts may be explained by involvement either of cerebral or spinal centers. In the independent cases, sensory and motor disturbances arise in the parts supplied by the affected segments of the cord. Pain and spasm or loss of power are most prominent.

Treatment is that of cerebrospinal meningitis.

CHRONIC SPINAL LEPTOMENINGITIS

Etiology.—Chronic inflammation of the spinal pia mater rarely results from the acute form, but follows syphilitic or tubercular lesions of the vertebræ or cord, or results from injury.

Morbid Anatomy.—The pia mater of the cord is thickened, opaque, adherent. Gummata or tubercles may be present. The vertebræ or cord show corresponding lesions.

Symptoms.—Pain, spasm or paralysis in the parts supplied by the affected segments of the cord results. Some of the symptoms produced by tubercular or specific disease of the cord or vertebræ are explainable in this manner, but the symptoms due to the lesion of the membranes are usually obscured by the more important affection of the bones or cord, and the diagnosis is that of the primary condition.

Treatment.—The underlying condition (syphilis or tuberculosis) must be treated. Counter-irritation, local applications of heat or cold, and spinal douches may be employed.

AFFECTIONS OF THE BLOOD-VESSELS AND CIRCULATION OF THE CORD

Hyperemia.—Congestion occurs as part of acute inflammation of the membranes or of acute myelitis, poliomyelitis and the like. It need not be considered separately.

Anemia.—In some cases of severe anemia, especially pernicious anemia, marked changes in the spinal cord are found. The symptoms of these spinal changes may precede or overshadow those of the anemia. The symptoms vary. They are usually limited to the lower extremities and are of several types. (1) Pain and paresthesia (numbness, tingling, etc.) are commonly present, either in feet or hands.

(2) Spastic. The patellar reflexes are increased, the muscles and gait are spastic, weakness or paralysis are marked.

(3) Ataxic. The reflexes are lost, the gait ataxic, and the picture is that of tabes dorsalis.

Embolism and Thrombosis.—These lesions may occur under the conditions which cause cerebral embolism or thrombosis. They are not commonly recognized.

ARTERIOSCLEROSIS of spinal vessels occurs as part of the general-

ized process. Of recent years the resulting disturbances of function have received much attention under the designation of intermittent claudication or limp. (Erb.) (See page 232.)

Hemorrhage into the cord (hematomyelia). Bleeding into the substance of the cord results most often from trauma, falls on the back or blows on the spine and the like.

Exposure to cold, over-exertion and excessive coitus are also possible causes. Such affections of the cord as myelitis, syringomyelia or tumors may be complicated by hemorrhage.

MORBID ANATOMY.—The hemorrhage may be large or small. Softening and destruction of the cord vary with the extent of the hemorrhage. Not infrequently multiple foci are found. In old cases the blood is absorbed and only pigmented areas found.

SYMPTOMS.—Sudden onset of pain followed by paralysis, and then loss of sensation affecting the lower extremities or the whole body up to the neck, are characteristic. Rapid improvement in the symptoms, caused by resorption, is also suggestive. Incontinence of urine and marked constipation are usually present. Bed sores and cystitis are frequent complications.

Gradual improvement usually follows and complete recovery is possible. The presence of faradic response in a muscle or muscles is hopeful, loss of it a sign of permanent paralysis. Death may result at once from extensive hemorrhages or in other cases follow sepsis from bed sores or cystitis.

Treatment.—Protracted rest with care of the bowels and bladder is essential. Asepsis must be observed in catherization. After six or eight weeks, electricity, massage, passive and then active movements are employed to restore muscular power.

CAISSON DISEASE

Definition.—A condition characterized by pain and paralysis, usually paraplegia, met with in caisson or tunnel-workers on too rapid exit from the high-pressure chambers.

Etiology.—In deep tunnels and caissons an artificial air pressure of several atmospheres is maintained to prevent the entrance of water. Under such pressure the blood becomes surcharged with gases. On the withdrawal of the pressure some part of these gases (O and CO_2) must be rapidly discharged. Under normal conditions such discharge occurs in the lungs. If the fall of pressure is suddenly induced, these gases may be liberated internally, especially in the closed spinal canal, with resulting damage to the cord. Venous stasis similarly caused may play a part in the process.

Indulgence in alcohol, heart or kidney disease, or illness of any kind predisposes to this affection.

Prevention.—Examination of all tunnel workmen to exclude the unfit, shortening of the hours of work under high pressure to four or even to three hour ''shifts,'' and installing exit chambers in which the

men are required to remain from fifteen to thirty minutes, while the pressure is gradually lowered, have greatly reduced the number of cases.

Lesions.—In recent cases congestion of the brain and spinal cord sometimes accompanied by minute lacerations is found; in long-standing cases areas of softening in the cord and the appearances of a myelitis.

Symptoms.—These may develop immediately on leaving the lock or only after three or four hours. Pains in the legs or abdomen, giving rise to the popular name of "the bends," usually mark the onset. Loss of power may or may not follow. In severe cases paraplegia with complete loss of power and sensation in the lower extremities results. Sudden death may occur. Headache, nausea, vomiting, tinnitus or deafness, and retention of urine may accompany the onset. The milder symptoms disappear quickly; the severer cases run the course of a severe myelitis. Death may result from bed sores or cystitis, or recovery with weakness or partial loss of power in the legs may be the outcome.

Treatment.—Return to the high pressure immediately may bring prompt relief from symptoms. Cabinets for this purpose are now provided. Otherwise the treatment is that of myelitis.

MYELITIS

(Transverse or General Myelitis)

Definition.—A localized degeneration or inflammation of the spinal cord involving the entire area of one or more segments.

Etiology.—The term myelitis covers a number of affections of the spinal cord, agreeing only in the fact that they affect the total area of some part of the cord. The etiology is therefore diverse. Transverse myelitis may follow: (1) Certain acute infectious diseases, especially the exanthemata, rheumatism, or septicemia. (2) The toxemia of pregnancy or the puerperium. (3) Syphilis in 75% of cases. Trauma, exposure to cold, and excessive sexual indulgence are regarded as contributory causes.

Morbid Anatomy.—Softening of some part of the cord is found, and it may be diffluent or even purulent. Hyperemia or small hemorrhages are found on the surface of the affected portion. On section the outlines of the gray and white substance are indistinct and the clear color and markings of the normal cord are lost.

Microscopically the appearances include hemorrhages, infiltration about the blood-vessels, and degeneration of the ganglion cells. The degenerations may be traced upward or downward in the several tracts of the cord. In old cases the cord becomes sclerotic, and the ascending and descending degenerations can be traced for some distance from the seat of softening.

Bacteriology.—Staphylococci, streptococci and pneumococci have been found in various cases. No one organism is constant.

Symptoms.—The onset may be sudden and acute with fever, or in children convulsions; or it may be gradual and insidious. Premonitory tingling, numbness, formication, hyperesthesia or girdle sensation may be felt.

The extent and distribution of symptoms will depend upon the level of the cord affected. If the lesion is in the sacral portion only the lower extremities will suffer. If in the dorsal region part of the trunk also will be involved, and if the lesion be cervical all the trunk and the arms, as well as the legs, participate in the disturbance. The motor symptoms are most impressive. Power is lost in part or altogether.

The legs, or both legs and arms, are partially or totally paralyzed. If the lesion is high, the diaphragm and respiratory muscles may be involved and dyspnea result. There is regularly difficulty in emptying the bladder and rectum, and retention and constipation, or incontinence from overflow result.

Sensory symptoms are less pronounced. Sensation is lost in part or altogether. At the level of the lesion there may be a narrow zone of hyperesthesia.

The deep reflexes are lost if the lesion involves the centers concerned in their production; they are increased if the lesion is above these centers. Ankle clonus and exaggerated patellar reflexes are therefore common. Babinski's sign is present.

Atrophy of the muscles occurs from disuse, and they become soft and flabby, but the electrical reactions are preserved. Rigidity may develop. Bed sores and cystitis are frequent and dangerous complications.

The duration of the affection varies from days to years. The condition tends to become chronic—the paralysis permanent with resulting contractures and deformities, the incontinence established, bed sores or other septic infection (cystitis) troublesome. The patients die of exhaustion or from intercurrent infection.

The course in some cases is swiftly fatal, in others life is prolonged for years. Recovery may occur in milder cases.

Diagnosis.—The combination of paraplegia with loss of sensation and loss of control of the sphincters is characteristic. Compression of the cord by tumors or fracture or dislocations of the vertebræ must be excluded.

Disseminated sclerosis must be excluded by the definiteness of the upper limit of the palsy and the absence of pupillary phenomena.

Functional paralysis (hysteria) must be considered, especially in young women. Loss of control of the sphincters, the presence of Babinski's sign, as well as absence of other signs of hysteria, must be conclusive.

Syphilis must be sought for in other parts. A Wassermann reaction is important. The cerebrospinal fluid should be obtained by lumbar puncture and examined for organisms and cells. A lymphocytosis favors the diagnosis of syphilis.

Treatment.—In early syphilitic cases specific treatment should be pushed, salvarsan followed by both mercury and the iodides.

In all cases, rest in bed, a reasonable diet and care of the bladder and rectum are essential. The catheter must be employed, if necessary, but under strict aseptic precautions. The bowels should be moved by laxatives or enemata. Careful nursing is essential to prevent bed sores.

After some weeks (six or eight) massage, electricity and passive motion may be employed for the paralyzed parts, and the patient should be encouraged to try movements for himself.

COMPRESSION OF THE SPINAL CORD

(Compression Myelitis)

Definition.—Interference with the functions of the spinal cord due to pressure.

Etiology.—The most frequent cause is caries of the spine, due to tuberculosis. The resultant "knuckling" of the spine and possibly the products of inflammation serve to compress the cord, and more or less completely abolish its functions. Children suffering from *Pott's disease* therefore often present symptoms of compression.

Aneurisms or other thoracic or abdominal tumors may erode the spine and compress the cord.

New growths of the spine itself or the membranes of the cord, abscesses, hemorrhages or gummata located within the spinal column, extreme lateral curvature, fracture or dislocation of the spine may have this result.

Morbid Anatomy.—The cause of compression is found in caries of the spine or some of the other causes mentioned. The cord itself is smaller than normal and softened at the affected part. The outlines of gray and white matter may be obliterated, and the substances of the cord may be fluidified or in late cases replaced by fibrous tissue.

Microscopically the cells of the cord are degenerate or absent, and above and below respectively in long-standing cases there will be degeneration of the ascending or descending tracts of the cord.

The SYMPTOMS, like those of a myelitis, depend upon the level of the cord affected. In the great majority of cases the compression is in the dorsal or lumbar region. Weakness of the legs, increasing gradually to complete loss of power, loss of control of bladder and rectum, and loss of sensation in the lower extremities usually develop. In cases of fracture or dislocation these symptoms come on suddenly. If the lesion is cervical, loss of power and sensation is seen in the arms, and there may be dyspnea and cyanosis.

At the upper margin of the area of anesthesia there may be a narrow band of hyperesthesia.

The reflexes, if their centers are involved, are lost; if the lesion is above these centers, they are exaggerated. Babinski's reflex should be present.

The course of the affection depends upon the cause. Cases due to Pott's disease may, under treatment, become quiescent or recover. If due to gummata, recovery follows specific treatment. In other cases the condition grows gradually worse, and the patient dies of exhaustion, bed sores, cystitis or pyelonephritis.

Treatment.—The cause must be treated. Pott's disease requires proper apparatus, and the measures applicable to tuberculosis in general. Rest in bed upon the back and extension are sometimes required.

Gummata demand the trial of salvarsan, followed by iodide of potassium in increasing doses, and mercurials. Otherwise the treatment must be wholly symptomatic. The danger of bed sores and cystitis should be remembered.

ACUTE POLIOMYELITIS

(Acute Anterior Poliomyelitis or Infantile Spinal Paralysis)

Definition.—An acute infectious disease characterized by acute inflammation and degeneration of the gray matter of the spinal cord, especially the anterior horns, and by early and persistent paralysis and atrophy of the muscles whose nerve supply comes from the affected parts of the cord.

Etiology.—The disease is endemic in certain regions, such as the Scandinavian Peninsula. In America it has occurred in repeated outbreaks involving more or less of the country. These outbreaks spread from ports of entry or from centers occupied by Scandinavian peoples. The disease is probably spread by persons who have come into contact with the sick, but who themselves have not had it. The infective agent, as in cerebrospinal meningitis, probably lodges in the nasal cavities, and thence reaches the brain and cord.

Children under five years of age constitute the great majority of victims, but older children or adults may suffer. The disease rarely involves more than one member of a family, and breaks out in most unexpected quarters. Susceptibility must therefore vary greatly. The children of both rich and poor are affected.

Most cases develop during the hot summer months, and cold weather checks the outbreaks.

Teething, exposure to cold, sitting on damp ground, previous acute infections, such as measles or scarlet fever, have been regarded as predisposing causes, but their influence is indeterminate.

Morbid Anatomy.—The cord is especially affected, but not infrequently in fatal cases the brain also is involved. The lumbosacral cord is most affected and the lesions lessen toward the brain. These lesions include (a) congestion of the meninges and of the affected section of the cord, (b) perivascular infiltration, masses of small round cells surrounding the vessels, and following them from the meninges into the substance of the cord, and (3) degeneration of the nerve-cells in the affected portions of the cord. The degeneration affects especially the ganglion cells of the anterior horns, but appears also in the posterior horns, and to a slight extent in the white matter of the cord.

Secondary degenerations are found in nerves from the affected segments of the cord, and the dependent muscles are markedly atrophic.

THE INFECTIVE AGENT.—Recent investigations show that the virus of acute poliomyelitis can be passed through the finest filter, and that thus far it has defied detection by the microscope. It can be inoculated from man to monkeys and from one animal to another by using emulsions of the central nervous system. It thus closely resembles the virus of hydrophobia, but the exact agent of infection is still unknown.

Symptoms.—The onset is often marked by sudden fever, 102° and 103° F., vomiting and prostration, or these symptoms are entirely lacking.

A day or two after the onset the paralysis appears, or the paralysis may be the first symptom. The paralysis involves one or both legs, or all four extremities, is usually complete at first, with flaccidity of the muscles and absence of reflexes. At the onset and for some time afterward the child may suffer pain, and cries when handled.

The fever, if present, lasts only a few days, and the constitutional disturbance quickly passes. The sensory disturbance usually disappears shortly, but the paralysis remains. This is, however, always much more extensive at first than later. Recovery of power usually appears in the arms in a few days, if they have been affected, later one leg shows improvement, then the other, and in rare instances the paralysis wholly disappears. A more or less extensive paralysis of one or both legs usually remains, and even the trunk or arm muscles may fail to regain power. With the paralysis atrophy occurs in the affected parts, and becomes extreme in the muscles which remain paralyzed. The reflexes return after a time in the course of recovery, but remain absent if the paralysis is permanent. The reaction of degeneration develops in the paralyzed muscles. Improvement in the electrical reactions, especially the return of faradic response, is significant of recovery.

The final outcome is permanent paralysis and atrophy in the muscles supplied from one or more spinal segments. The sensory symptoms always disappear early. The permanent paralysis is partial or complete, affects most often the tibial or peroneal muscles, causing permanent drop-foot, but often involves the muscles of the thigh, and occasionally the trunk as well. If the thigh muscles are very weak the patient cannot walk without support to the knee. If the trunk is involved, the patient cannot sit up without support.

The paralyzed limb is cold and thin, the muscles are atrophic, the ligaments relaxed, so that joints are abnormally movable (flail-leg). Secondary contractures occur and pes cavus, talipes-equino-varus or valgus, and contractures at the knee or hip are common. The affected limb fails to grow normally, and gradually becomes shorter than its fellow.

The duration of improvement is uncertain. Most of it is seen within three months; after a year or eighteen months there is little hope for muscles still paralyzed. The reflexes and electrical relations are the

best guides to recovery. Although regaining no new power, children often make great progress in walking, getting about when supplied with adequate apparatus.

LUMBAR PUNCTURE at the onset shows an increased amount of spinal fluid containing an excess of lymphocytes. No organisms are discoverable.

Prognosis.—Death from acute poliomyelitis is very rare; entire recovery is almost as unusual. A persistent paralysis of one or more groups of muscles is the rule.

Treatment.—The patients should be isolated. In cases of epidemics the schools should be closed. The acute stage has almost always passed before the diagnosis is made. Rest in bed, a light diet, a mild purge and care to prevent retention are indicated. Pain may require relief by aspirin, phenacetin or opium. For the paralysis, the limbs should be kept warm, passive movements practiced to prevent contractures, gentle friction employed, and later massage and electricity. The patient should be encouraged to practice and employ every movement possible. Later orthopedic apparatus is of great help, and in some cases grafting of nerves or tendons has been attended with success.

ACUTE ASCENDING PARALYSIS
(Landry's Paralysis)

Definition.—An acute paralysis beginning in the legs and rapidly involving the trunk and upper extremities, usually fatal, and not characterized by definite lesions of the cord.

Etiology.—Men between the ages of 20 and 40 years are most often affected; otherwise the etiology is unknown.

Morbid Anatomy.—In his original cases Landry found no lesions of the cord, and the absence of gross or easily discernible lesions may be regarded as characteristic. Delicate microscopic changes, not discoverable by the methods of fifty years ago, may, however, be found in the spinal cord, such as breaking up or loss of the chromatin granules in the nerve-cells of the anterior horns and Clarke's columns, and fatty degeneration of the myelin sheaths of the main spinal nerves, possibly of the peripheral nerves.

Symptoms.—The onset is marked by a sudden weakness, followed by paralysis of the legs. The muscles are flaccid. The muscles of the trunk, arms and neck are involved in turn. As the paralysis ascends, the respiratory muscles become involved, dyspnea and cyanosis result, and death usually ensues. In a few cases loss of power in the muscles of the palate, tongue and face has been observed. Apart from the rapidly advancing paralysis, the picture is negative. The reflexes are lost, but the muscles do not atrophy and the electrical reactions are preserved. There are no sensory disturbances of note, and the sphincters are not affected.

The duration of the disease is from two days to two weeks in fatal

cases. If recovery occurs convalescence may be protracted. Broncho-pneumonia may complicate the course.

Diagnosis.—Landry's paralysis is often confused with anterior poliomyelitis and acute multiple neuritis. From anterior poliomyelitis, the absence of other cases, the age of the patient, the absence of constitutional disturbance, the non-appearance of muscular atrophy and the preservation of the electrical reactions may serve to distinguish it. Post mortem the characteristic lesions of anterior poliomyelitis are not found.

From acute multiple neuritis distinction must rest on the knowledge of a cause (such as alcoholic or metallic poisoning, etc.), the presence of sensory disturbances, such as pain, tingling, numbness or anesthesia, and muscular sensitiveness to pressure. The rarity of involvement of the trunk in neuritis (preservation of the abdominal reflex), and the loss of electrical reactions are significant.

Landry's paralysis is symmetrical and complete. Multiple neuritis may be unevenly distributed, and some muscles may be spared.

Prognosis.—The possibility of recovery must be admitted, but the usual outcome is death within a few days.

Treatment.—There is no effective treatment. Rest in bed, a light diet, and regulation of the bowels are rational measures. Catheterization may be required. For respiratory embarrassment atropine and strychnine should be given. Frequent changes of position to prevent pulmonary congestion are advisable. If the patient survive, the treatment must be that of anterior poliomyelitis.

PROGRESSIVE MUSCULAR ATROPHY
(Chronic Anterior Poliomyelitis)

Definition.—A progressive atrophy of muscles, usually associated with rigidity and paralysis, and due to degeneration of the motor neurons of the cord.

Etiology.—The disease belongs to adult life and to men rather than women (five to one). Beyond these facts the causation is unknown.

Morbid Anatomy.—The cord appears normal, but microscopic examination shows progressive atrophy in the cells of the anterior horns and the central gray matter of the cord. The cells become small and shrunken, and many disappear. There are no signs of inflammation or degeneration. The axones and dendrites of these neurons likewise shrink and disappear. In the affected muscles there is simple atrophy of the muscle fibers. The atrophic process may be found at any level of the cord. It is usually most marked in the sacral region.

Symptoms.—The disease is chronic in type, lasting many years. The affected muscles become weak, then paralyzed, and more or less rigid. Fibrillary twitchings and cramps are common in them. There are no sensory or trophic disturbances. Several types of the disease depending upon the parts of the cord affected are described.

1. Peroneal type. The paralysis begins in the peroneal muscles, then affects the anterior tibial group. Weakness in walking and drop-foot result. Gradually the process advances into the muscles of the thigh, the adductors and glutei. Walking becomes more difficult and going up-stairs or rising from a chair may be impossible. The weakness and atrophy go steadily on till walking can no longer be accomplished. Sensation is normal. There is no edema, nor bed sores. Bladder and rectum are unaffected.

2. Ascending type. (Duchenne.) The muscles of the trunk are also involved. The patient cannot sit upright. The hands and shoulders become affected, as in the following type, and the condition ends in bulbar palsy.

3. Aran-Duchenne type. The paralysis and atrophy begin with the muscles of the thenar and hypothenar eminences of the hand. Weakness is felt in writing, in buttoning garments and the like. Later the lumbricales and interossei atrophy and the whole hand appears thin.

Contractures give the hand the attitude of "main en griffe" or of claws. One hand is affected after the other, then atrophy appears in the shoulders and the deltoid, biceps, brachialis anticus and other shoulder muscles are involved.

Some muscles escape the atrophy, and these by contrast appear hypertrophic.

Finally the intercostals and other muscles of respiration are involved, and respiration becomes wholly diaphragmatic.

The terminal stage is marked by involvement of the muscles of the face, tongue and throat, giving rise to bulbar paralysis (q. v.). The patients usually die of intercurrent disease. Life may be protracted for many years (two to twenty-five).

The muscles gradually lose their electrical reactions, and in the end make no response to either galvanic or faradic currents. The reflexes are similarly lost as the muscles concerned atrophy.

Diagnosis.—Progressive muscular atrophy must be distinguished from the following:

1. Amyotrophic lateral sclerosis, by the presence of exaggerated knee-jerks, Babinski's reflex, and increased tendon reflexes in the arms; also by some spastic rigidity in both arms and legs. The progress in this condition is more rapid.

2. Muscular dystrophy, characterized by appearances of hypertrophy, a different grouping of the muscles affected, and the absence of fibrillary twitchings.

3. Multiple neuritis, marked by sensory symptoms, tenderness along the nerve-trunks, short and rapid course, and some definite causation, such as lead or alcoholic poisoning.

4. Syringomyelia, early presenting like symptoms, but later developing characteristic dissociation of sensation (loss of response to heat and cold, with maintenance of pain sense), and trophic changes.

The PROGNOSIS is always unfavorable, but life may be prolonged indefinitely.

Treatment.—The general nutrition must be maintained by attention to diet, fresh air, and general hygiene. Massage and rubbing may be employed. Electricity appears to be useless. Strychnine and arsenic may be given as tonics.

GLOSSO-LABIO-LARYNGEAL PARALYSIS

(Progressive Bulbar Palsy)

Definition.—A progressive paralysis and atrophy of the muscles of the tongue, lips and larynx.

Etiology.—The disease commonly develops in late adult life, apparently as the result of exhaustion of the motor centers of the pons. A few early cases, probably due to congenital deficiency, have been recorded. No satisfactory explanation of the disease has been given.

Morbid Anatomy.—Degeneration of the nuclei of the hypoglossal, glossopharyngeal, pneumogastric and spinal accessory nerves is marked. Many of the cells disappear. In rare cases the nuclei of the seventh and fifth nerves are involved. The lesions of amyotrophic lateral sclerosis are often associated; some writers say they are always present. Atrophy of the muscles of the lips, tongue, pharynx and larynx can be shown.

Symptoms.—A slowly progressive loss of power in the affected muscles is shown by difficulty in speech, in mastication and deglutition, in the movements of the tongue, in swallowing and phonation. Difficulty is first noted in the pronunciation of the labials and dentals p, b, l, m, n, d, g, k, v, s, t. The lower lip droops and saliva escapes. Food collects between the lips and the gums, chewing becomes difficult and swallowing impaired, so that fluids regurgitate through the nares. The tongue cannot be protruded, and finally the voice fails. If the facial nerve is involved, the face loses expression. Involvement of the pneumogastric causes dyspnea and rapid heart-action. The patients become emotional, laughing or crying on slight provocation.

Atrophy and fibrillary tremor appear in the lips and tongue. The affected muscles give the reaction of degeneration. Death after one or two years results from inanition or bronchopneumonia (aspiration).

Diagnosis.—The combination of atrophy, fibrillary tremor, and paralysis of the lips, tongue, pharynx and larynx, and slow, progressive course are characteristic. The signs of amyotrophic lateral sclerosis, exaggerated reflexes, spasticity and weakness in the extremities are usually present. Bulbar tumors or hemorrhage do not produce symmetrical lesions. Myasthenia gravis shows no atrophy, no reaction of degeneration, and displays the characteristic early exhaustion on effort.

A pseudo-bulbar paralysis may result from lesions of the facial centers in the cortex or the motor neurons leading from them (internal capsule), but without atrophy, tremor or degeneration.

Bulbar palsy may form the terminal stage of either amyotrophic

lateral sclerosis, locomotor ataxia, syringomyelia, or chronic anterior poliomyelitis.

Treatment.—Attention to the diet, giving such easily digestible foods as can be swallowed, or feeding by gavage, is of help. No known treatment influences the underlying process or checks the disease.

LOCOMOTOR ATAXIA

(Tabes Dorsalis. Posterior Spinal Sclerosis)

Definition.—A chronic disease of the sensory neurons resulting in incoördination, sensory and trophic disturbances.

Etiology.—Locomotor ataxia is a disease of adult life, of men rather than women, a sequel of syphilis in from 50 to 90% of all cases. Traumatism, such as blows or falls on the back, exposure to cold or wet, or exhaustion from overexertion or underfeeding are predisposing causes.

Morbid Anatomy.—The lesions are found in the ganglia of the spinal nerves and in the fibers leading from them into the spinal cord. They begin and are most marked in the lumbar segments of the cord and develop later, and to a less extent upward. The lesions consist in a degeneration of the nerve-fibers and a secondary increase in the interstitial tissue. Theoretically the lesions of the ganglia should precede those of the nerve-fibers leading from them, but this is not always the case, and the sclerosis of the fibers leading from the ganglia into the cord —that is, the posterior columns—is the most pronounced feature of the lesion. The columns of Goll and Burdach are converted into dense connective tissue with degeneration and disappearance of the nerve-fibers.

The lesions may be more widespread and involve the columns of Lissauer and Clark. In rare cases the process progresses upward, and the ganglia of cranial nerves, the hypoglossal, glossopharyngeal, the facial and even the fifth nerve have been found involved.

In still rarer instances the sclerosis spreads into the anterior columns of the cord, producing motor symptoms.

Symptoms.—The cardinal symptoms of locomotor ataxia include: (1) Pains and paresthesia, usually in the legs. (2) Bladder disturbances. (3) Ataxia of the lower extremities, rarely of the upper. (4) Girdle sensation. (5) Sexual weakness. (6) Weakness of vision or double vision. (7) Visceral crises. (8) Trophic disorders.

The cardinal physical signs are: (1) Loss of deep reflexes, especially the patellar and Achilles reflexes. (2) Argyll-Robertson pupil, reaction of the pupil to light, but not to accommodation. (3) Romberg's sign, swaying of the body when the feet are put together and the eyes closed. (4) Atrophy of the optic nerve or paralysis of ocular muscles. (5) Areas of anesthesia, corresponding to certain spinal segments.

The evidences of the disease develop slowly in the course of months or years. The exact grouping of the symptoms and signs varies in

different cases. For convenience of description the course may be described in stages.

1. Preataxic stages. Paresthesia and pain in the lower extremities in most cases mark the onset. Tingling, numbness, burning, formication, sensations of cold, or of walking on cotton or on pins, may be complained of. Pain is usually sharp, lancinating in character, lightning-like, either deep or superficial, and occurring in attacks.

The girdle sensation is a form of hyperesthesia leading to the sensation of a band or girdle tied about the body. As the disease advances upward the level of the girdle or band rises, and it may be felt about the neck.

Attacks of pain in other parts of the body than the legs are not infrequent, and are associated with disturbances which are commonly known as visceral crises. Gastric crises, consisting in sudden attacks of pain in the epigastrium, accompanied by nausea and vomiting, unrelated to the taking of food, are well known. Such attacks last for hours or days, and are relieved only by morphine. Laryngeal crises in which the patient is seized by sudden cough, attended with dyspnea (due to adductor spasm) and a sense of severe constriction of the throat, are not infrequent. Vesical, urethral, intestinal and rectal crises have also been described. Such crises may be the earliest symptoms of locomotor ataxia.

The eye symptoms may be the first of the disease, the patient complaining of gradual loss of sight, of diplopia, or of strabismus.

Examination at this stage usually discloses the absence or impairment of the patellar and Achilles reflexes, the Argyll-Robertson pupil, and Romberg's sign. But in the early stages one or other of these signs may be missing—while the presence of some of them is essential to the diagnosis. In the ocular cases, atrophy of the optic nerve, or paralysis of some of the eye muscles is present.

2. Ataxic stage. After more or less pain, or some of the other initial symptoms have been present for some time, ataxia appears, usually in the legs, sometimes in the hands. This ataxia is due partly to anesthesia, partly to loss of muscular sense, and possibly to loss of sensation from the joints. The patient notes that when he closes his eyes, as in washing his face, or when he walks in the dark, he loses his balance. Romberg's sign is closely related to these disturbances. The gait now becomes characteristic. In walking the foot is thrown outward and then forward with exaggerated motion and brought down with a slap on the floor. The whole action is exaggerated and awkward. The feet are widely separated and the steps are unequal and uncertain. The patients maintain their balance and walk only with the aid of their eyes, and if sight is excluded they stagger or fall. This ataxia can be brought out by asking the patient to follow a line with his toe, to cross the knees, to touch one heel to the opposite knee, etc.

Ataxia may appear in the hands, and awkwardness is shown in writing or in buttoning the clothes. The movements of fingers and hands

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become exaggerated and uncouth. In the late stages the simplest movements may be executed with difficulty. Loss of the sense of weight occurs at this time, and the patient becomes unable to locate the position of his limbs when deprived of sight.

With the ataxia sensation becomes blunted and then lost. Pain and temperature sense both suffer. The sense of pain may be much delayed or lost. Temperature sense is variously affected, sensibility to cold often being retained when that to heat is lost. On account of this loss of sensibility these patients may easily be burned or injured without their knowledge.

AREAS OF ANESTHESIA limited to the inner or outer aspects of the legs or more general in distribution may be shown. These anesthetic areas correspond not to the distribution of peripheral nerves, but to the segments of the cord affected.

TROPHIC DISTURBANCES develop. Herpetic eruptions appear on the legs or body. A perforating ulcer may develop on the sole of the foot. The nails become ridged or fall out; the teeth may be lost. Certain joint lesions (Charcot joints) are especially striking. One or more joints, most often the knees, become enlarged and deformed with much thickening about the joints and possibly effusions into them, with some disability, but little or no pain. The joints may suppurate. The muscles may become atrophic. Relaxation of muscles and ligaments allows of excessive movement, especially at the knee or hip, the hypotonia of Fraenkel.

Mental symptoms are seen late in some cases. Mania, melancholia or paresis may develop. In some cases the mental disturbance is transitory.

3. Paralytic stage. The patient finally becomes unable to walk, is bedridden, helpless. Bulbar paralysis may appear in this stage. Deaths occurs from cystitis or resulting pyelonephritis, from exhaustion, or from intercurrent disease, rarely from locomotor ataxia itself.

Course and Prognosis.—The disease is slowly progressive—covering a period of ten or twenty years. It may become stationary at any stage, and recovery is possible, although most rare.

Diagnosis.—The emphasis in diagnosis must be put on the signs rather than symptoms, the loss of reflexes, Argyll-Robertson pupil and Romberg's sign especially. The presence of one or more of these signs with any of the symptoms justifies a diagnosis of tabes dorsalis.

Multiple neuritis is distinguished by the rapidity with which symptoms develop and improvement occurs, by involvement of both arms and legs with integrity of the bladder and rectum, the difference in the areas of anesthesia (corresponding in neuritis to the peripheral nerves, and hence being stocking or glove-like, while in tabes they correspond to spinal segments and cover long areas on one or the other aspect of the limbs) and the absence of pupillary changes.

General paresis may in the early stages present symptoms of tabes, or late in tabes evidences of general paresis are present. The order of

development and duration of the symptoms will then be important. Observation for some time may be required.

Cerebellar disease may cause ataxia, but the incoördination disappears if the patient lies down, and headache, vomiting, etc., are usually present.

Neurasthenia. The reflexes can be obtained by diverting the patient's attention or suddenly tapping the tendon, and the pupillary signs are absent.

Ataxic paraplegia is identified by the spasticity of the muscles and the increase of reflexes.

Syphilitic meningitis of the cord develops rapidly, and causes pain in the back; the knee-jerk is not lost, and the pupil reacts to light.

The spinal fluid regularly shows an increased cellular content, mainly lymphocytes. It may also give the Wassermann reaction.

Treatment. General.—Residence in a mild, equable climate is advantageous. The diet should be nutritious and suited to the patient's digestive power. Exercise must be carefully regulated to avoid exhaustion, and yet the patient should be encouraged to keep up and about rather than to take to bed. The condition of the bladder must be watched and the catheter employed, if retention occurs. The dangers of infection must be borne in mind and strict asepsis practiced. For constipation laxatives or enemata should be employed.

Hydrotherapy in the form of tepid douches to the spine, the temperature varying from 75° to 95° F., is of value. Tub or sitz baths of like temperature may be employed. Extremes of heat or cold are harmful.

Specific treatment for all syphilitic cases should be thoroughly carried out, salvarsan being tried, and later mercury and the iodides given to the point of tolerance. Inunctions of mercurial ointment, ½ dram daily, or the protiodide may be given in doses of gr. 1/6 to gr. ⅓ thrice daily. The iodide is given in increasing doses up to 30 to 60 grains, three times a day. The Wassermann reaction is useful as an indication of the need and duration of specific treatment.

In other cases arsenious acid or bichloride of mercury, in doses of gr. 1/50 of either, three times a day, may be employed.

For the pains, analgesics, phenacetin, acetanilid or aspirin, may be given. In severe attacks, such as the crises, morphine is required.

For the ataxia the educative exercises of Fraenkel are employed. In these the patient practices following a straight or curved line with his toe, taking steps of measured length, following the sides of a triangle, etc. These exercises are repeated several times daily. Gradually the patient advances to walking upstairs and more difficult movements. Patient practice improves the use of feet or hands.

LATERAL SCLEROSIS

Definition.—A progressive paresis and rigidity of the muscles, usually beginning in the legs, without sensory symptoms or atrophy, due to a sclerosis of the pyramidal tracts.

Etiology.—The pyramidal tracts being made up of the axones of motor neurones lying in the cerebral cortex, a descending degeneration and secondary sclerosis result from any injury to those neurones or any interruption in their course. The following lesions may be found:—

1. Cerebral lesions, such as hemorrhages, tumors or chronic inflammation. 2. Spinal lesions, such as transverse myelitis, hemorrhage or tumors. 3. Disease or injury of the spine, such as caries, tumors or pachymeningitis, compressing the cord. Secondary lateral sclerosis is therefore frequently seen.

A primary lateral sclerosis is established, but the etiology is obscure. The affection occurs between the twentieth and fortieth year. Syphilis is occasionally the cause.

Symptoms.—A progressive disability with stiffness and a tendency to cramps and tremors in the legs constitute the essential features of the disease. The beginning is insidious and the progress slow. The patient early notes a slight difficulty in moving the legs. The difficulty may first appear on one side, but soon both legs are involved. Walking, going upstairs, running or any movement is attended with increasing difficulty. The muscles become rigid, cramps are frequent, and passive motion of the legs meets with increasing resistance, especially in the adductors. The tendon reflexes are exaggerated. Ankle clonus and Babinski's reflex are present. There are no sensory disturbances of note, no atrophy, no change in electrical reactions. The loss of power slowly increases. The gait becomes characteristic. The patient takes short steps, throwing one foot around the other on account of the tendency to overlapping due to adductor spasm, the weight is borne on the ball of the foot and the heel scarcely touches the floor. Clonus may be excited by the touch of the toes on the ground. Gradually the power of walking is lost and the patient becomes bedridden. The legs then become flexed until the heels may touch the buttocks, and so rigid that they cannot be drawn down. The slightest touch excites reflex contraction and tremors in the muscles. Atrophy from disuse may follow.

The condition is very chronic, enduring for twenty or thirty years. Death results from intercurrent disease. Even in advanced cases the hands are rarely affected.

Diagnosis.—The symptoms and signs of lateral sclerosis are easily recognized. That the disease is primary can be affirmed only by the exclusion of the many causes for the secondary condition. Hysteria must be excluded.

TREATMENT of the primary type is wholly palliative. Division of the posterior roots of the spinal nerves supplying the affected muscles has been employed with some relief. In the secondary cases the primary condition must be treated.

HEREDITARY SPASTIC SPINAL PARALYSIS

Definition.—A spastic spinal paralysis, occurring in families, due to lack of development or degeneration of the pyramidal tracts.

Etiology.—Beyond the fact that the affection develops in several members of a family, nothing of value is known.

Morbid Anatomy.—Degeneration of the pyramidal tracts throughout the cord, but especially in the lumbar region, is found. Occasionally the degeneration involves neighboring tracts, such as the direct cerebellar and the column of Goll.

Symptoms.—These are those of lateral sclerosis, as given above. The symptoms appear in the first year or in adult life. Some patients develop symptoms rapidly, some slowly. The hands are rarely affected. The familial occurrence is the only distinctive feature of these cases.

Diagnosis.—Secondary lateral sclerosis must be excluded. The occurrence of other cases in the family is the only clue to the hereditary nature of the affection.

TREATMENT is that of lateral sclerosis.

ATAXIC PARAPLEGIA

Definition.—A sclerosis of the posterior and lateral columns of the cord, producing spastic paraplegia with ataxia.

Etiology.—Middle-aged men are most often affected. Injury or exposure may precede the onset.

Morbid Anatomy.—A sclerosis of the lateral pyramidal and the adjacent portions of the posterior columns is found.

Symptoms.—The patient suffers from progressive weakness in the legs, together with unsteadiness of gait and lack of coördination in the movements remaining. No pain or other sensory symptoms develop. The reflexes are exaggerated, and the spasticity, cramps and tremor of lateral sclerosis appear. The sphincters are not involved and the eye symptoms appear late.

FRIEDREICH'S ATAXIA
(Marie's Hereditary Cerebellar Ataxia)

Definition.—A combined sclerosis of the posterior nerve-roots, the posterior and lateral columns, producing ataxia and paraplegia.

Etiology.—The disease occurs in several members of a family, apparently owing to some congenital defect. Alcoholism, tuberculosis, or syphilis in the parents predisposes to it. A neuropathic tendency shown by the occurrence of migraine, epilepsy, hysteria, or insanity is often found in the family. Trauma of any kind, an acute infectious disease, or any influence lowering the vitality of the patients favors the development of the disease.

Morbid Anatomy.—The cord is smaller than normal, the atrophy affecting especially the posterior columns. On section a sclerosis is found involving the columns of Goll and Burdach, the direct cerebellar tract, and the antero-lateral tract of Gowers. The pyramidal tracts, especially the crossed, are affected in some degree. The cells in the column of Clarke are degenerate.

Symptoms.—The disease appears early, before puberty as a rule. Ataxia of the lower extremities is first noted. The child walks with feet wide apart, bringing the foot down with a stamp, balancing himself by the aid of the hands, like an intoxicated person. Romberg's sign may be present. The knee-jerks are lost; Babinski's reaction present. Gradually the ataxia affects the upper extremities, leading to awkward movements, suggesting chorea. Swaying movements of the body and head develop. The muscles of the trunk are weakened and the spine appears scoliotic.

Contractures of the muscles of the extremities produce talipes equino-varus and other deformities. Speech becomes slow and jerky and nystagmus is present. The mind remains clear. The muscles of the extremities are atrophic and electric excitability is diminished.

Cerebellar type. Marie and others have described a cerebellar type in which with the ataxia the patellar reflexes are retained, the legs become spastic. Lancinating pains may occur in the early stages, but sensory or trophic disturbances are absent in both.

Course.—The disease is progressive, the patients are finally bed-ridden, but may survive twenty or thirty years.

Treatment is that of locomotor ataxia.

SYRINGOMYELIA

Definition.—A chronic disease of the spinal cord, characterized pathologically by the presence of one or more abnormal cavities in the cord, and clinically by dissociative anesthesia, trophic disturbances and progressive muscular atrophy and paralysis.

Etiology.—The disease develops between twenty and forty years, in men more often than in women. The patients often present defects of development, such as infantilism, bosses on the skull and spina bifida occulta. Trauma of the head or spine may precede the onset.

Morbid Anatomy.—The essential feature is the presence in the cord of one or more pathological cavities. Externally the cord appears normal, or may be enlarged and fluctuating in some part, if the cavity is large. On section the cavity is found, usually in close relation to and communicating with the central canal, more often involving the posterior parts of the cord. The cavity is rarely symmetrical, one side being, as a rule, decidedly more affected, but the cavity may be of any shape or size, so that it may replace almost the whole area of the cord, leaving only a shell-like remnant.

The length of the cavity also varies greatly, involving in some cases only two or three segments, and in others the whole length of the cord. The cavity rarely extends into the brain, but has been seen to extend to the fourth ventricle, or even the internal capsule.

The contents of the cavity are clear serum; its wall is made up of thickened neuroglia tissue. In some parts the cavity is lined with epithelium apparently from the central canal, with which the cavity often communicates. If a large area of the cord is involved there may

be secondary ascending and descending degenerations from the level of greatest destruction.

The pathogenesis of syringomyelia is thus far undetermined. Several explanations are offered. (1) Congenital defects of development. (2) A primary increase in neuroglia tissue (gliosis) due to inflammation, with secondary degeneration of the cord. (3) Interference with circulation due to vascular changes, which determines the degeneration. (4) Intramedullary hemorrhage with like result.

Fig. 104.—Atrophy and anesthesia in syringomyelia, front view. (International Clinics, Vol. II, Series 20, page 219.)

Symptoms.—The patient complains of increasing weakness and atrophy in some part, usually the hand and arm, or of pains and paresthesia (tingling, numbness, heat or cold).

The characteristic conditions are found on examination, and include: (1) Dissociative anesthesia, the sense of touch being preserved while those of pain and temperature are lost. (2) Trophic disturbances of greater or less extent. (3) Atrophy and paralysis of some muscles. With these may be combined many other disturbances of the functions of the cord, depending upon the extent of the involvement of its sub-

stance. The evidences of syringomyelia are usually most marked on the upper extremities and trunk, but may first appear on the lower. Both sides are commonly involved, but one to a greater extent than the other. The areas of anesthesia, paralysis or atrophy while overlapping do not correspond to one another. If the entire area of the cord is involved at any level, the evidences of lateral sclerosis in the cord below that point appear. (See Figs. 104 and 105.)

Fig. 105.—Atrophy and anesthesia in syringomyelia, back view. (International Clinics, Vol. II, Series 20, page 219.)

1. **Sensory Disturbances.** (a) Thermo-anesthesia is almost constant. Sensation to both heat and cold is usually lost, but one may be preserved. The area involved may be small or large, and corresponds to certain spinal segments, not to the distribution of peripheral nerves. (b) Analgesia is usually present with thermo-anesthesia. (c) Tactile sensibility is long preserved, but may be lost in the final stages. (d) Pain or paresthesia may mark the onset. The pain may be severe.

2. **Motor Disturbances.** (a) Paresis or paralysis ordinarily appears in the hand. Atrophy accompanies it. The muscles of the hand or arm may be much wasted. Contractures may give the appearance

of ''main en griffe.'' The paralyzed muscles may show fibrillary tremors and the reaction of degeneration. (b) Spastic paralysis, loss of power without atrophy or degeneration and with increased reflexes, may appear below the level of most marked cavity formation. This spastic paralysis naturally appears most often in one or both legs, but may involve the upper extremities as well. (c) Spinal curvature or scoliosis, resulting from muscular inequalities, is quite common. It is most marked in the dorsal portion of the column. (d) Ataxia may be quite marked.

3. Trophic Disturbances. (a) Skin. Atrophy of the skin with eruptions of vesicles or bullæ is common. The skin becomes glossy, thin and cracks or fissures appear. Ulceration may follow, necrosis of bone develop and whole parts (fingers or toes) be destroyed. The nails are thickened and brittle; they may drop off. (b) Bones. Necrosis occurs in some cases. Hypertrophy or atrophy and spontaneous fracture are occasionally seen. (c) Joints. Arthropathies are frequent (25% of cases). The shoulder or elbow is most often involved; other joints may suffer. The changes are the same as those seen in locomotor ataxia (Charcot joints), thickenings, deformity, effusions, with destruction of the articular surfaces or the heads of the bones in some cases. These arthropathies are usually painless.

4. Vasomotor and Secretory Disturbances. Pallor or hyperemia or cyanosis, dryness or excessive sweating is frequently observed.

5. Accessory Disturbances. (a) Ocular symptoms. Owing to frequent involvement of the last cervical and first dorsal segments, pupillary and ocular symptoms are seen in the great majority of cases. The pupil is smaller, the palpebral fissure narrower and the eyeball is somewhat retracted. These affections give a suggestion of ptosis, which is promptly proven false, when the patient looks upward. (b) Bulbar symptoms appear late in the disease. Atrophy and tremor of the tongue, paralysis of vocal cords, difficulty in swallowing or in respiration may occur. (c) Sphincteric control. In the usual (dorsal) type of the disease, disturbance appears late. If the lumbo-sacral cord is affected at the outset, these disturbances appear early.

Course.—The disease is usually far advanced when recognized. It progresses slowly, but surely. Death after some years occurs from cystitis and its complications, from exhaustion due to bed sores and suppuration, or suddenly from rupture of the cord.

Diagnosis.—Syringomyelia in typical form is easily recognized, but easily overlooked in irregular types. The diagnosis depends mostly on thorough examination, which elicits the analgesia and disturbances of thermic sensibility. These and the trophic disorders distinguish the disease from chronic anterior poliomyelitis, progressive muscular atrophy or amyotrophic lateral sclerosis.

The asymmetry of the lesions of syringomyelia and the preservation of the sense of touch distinguish it from myelitis, transverse or disseminated. Tumors of the cord may give a like history, but regu-

larly cause severe pain, are steadily progressive, are limited to two or three segments of the cord, and cause symptoms of a transverse myelitis below the level of location.

Prognosis.—The disease is incurable, but very chronic. It often becomes stationary. TREATMENT must be symptomatic. Care must be taken to avoid burns or injuries. Paralysis must be treated as in anterior poliomyelitis.

MORVAN'S DISEASE is a type of syringomyelia in which trophic changes are very prominent. Pains and atrophy develop in the hands, with analgesia and anesthesia, and are followed by painless felons and possibly necrosis of the phalanges.

TUMORS OF THE SPINAL CORD

Tumors of the cord are rare. They may be of almost any variety. Sarcoma, tubercle, gumma, fibroma, glioma, are the more common forms. These growths may spring from the cord (intramedullary) or from the dura covering it (extramedullary). It must be recalled that compression of the cord is much more frequently the result of pressure due to caries of the vertebræ, or tumors growing in the vertebræ (sarcoma, carcinoma, myeloma), than of the pressure of tumors of the cord itself.

Etiology.—Tumors of the cord are more common in adult life, but may develop in childhood. (One in three under the age of sixteen.) Except for syphilis or tubercle, we can give no satisfactory causation.

Morbid Anatomy.—The tumors are of great variety. The most common site is the dorsal cord, but any part may be involved. Apart from the tumor itself the cord shows the effects of compression, or a myelitis may be set up.

Symptoms.—These are caused by pressure upon the cord, gradually increasing till the functions of the cord at the level of compression are destroyed. The symptoms are those of compression (page 505), but with certain fairly distinctive features. Pain is severe, neuralgic in character, intense, at first unilateral and sharply localized, but after a time spreading to the other side. The painful area is hyperesthetic.

At the level of the tumor there are atrophy and paralysis of muscles with reaction of degeneration, anesthesia of the skin, herpes zoster and trophic changes. Dissociative anesthesia (preservation of touch with loss of sensibility to pain and temperature) is suggestive of an intramedullary growth.

Below the level of the growth weakness with exaggerated reflexes, imperfect control of bladder and rectum, loss of sensation up to the level of the lesion, and a tendency to bed sores are developed.

The course varies with the nature of the tumor, but the average duration of life is less than two years.

Tumors of the cauda equina cause severe sacral pain and unusually widespread pain in the nerves of the legs. Paralysis and atrophy may be limited and are rarely symmetrical. Paralysis of bladder and rectum occurs early.

Diagnosis.—Caries of the spine from any cause must be excluded. Rigidity of the spine, tenderness over the spinous processes, increase of pain on motion or walking, and kyphosis must be sought for. A radiograph of the spine is valuable. The nature of the tumor is difficult to determine. Fever and other constitutional disturbances, and evidences of disease elsewhere, are found in tuberculosis. In syphilis the history is important, the presence of other lesions, or a Wassermann reaction helpful. An antecedent malignant tumor is suggestive. Otherwise we can only hazard a guess at the nature of the tumor.

Prognosis is unfavorable. Surgical removal of the tumor may be successful.

Treatment.—In specific cases active anti-syphilitic treatment is indicated. In other cases if the tumor can be reasonably localized, operation should be undertaken, unless the tumor is known to be malignant.

DISEASES OF THE BRAIN AND MENINGES
DISEASES OF THE MENINGES
EXTERNAL PACHYMENINGITIS

Definition.—An inflammation of the external layer of the dura mater.

Etiology.—Fractures or disease of the bones of the skull (caries following middle-ear disease and the like), syphilis, or tumors involving the bones, may be the cause. Erysipelas occasionally causes an acute inflammation.

Morbid Anatomy.—The dura is thickened, unusually dense and adherent. An accumulation of pus or blood may be found between it and the bone.

Symptoms.—The condition is often unexpectedly found at autopsy, especially in the insane. The symptoms are those of the primary trouble, with persistent headache and local tenderness.

Treatment.—Syphilitic disease should receive specific treatment. Otherwise the treatment must be directed to the cause. If pus is present, trephining must be done.

INTERNAL PACHYMENINGITIS

Definition.—An inflammation of the internal surface of the dura mater.

Etiology.—It may be part of an external pachymeningitis and arise from the causes of that condition. It is common in the insane. Sunstroke, alcoholism or syphilis and various wasting chronic diseases are regarded as causative.

Morbid Anatomy.—The dura is thickened and its inner surface is covered with a layer or layers of delicate connective tissue unusually rich in blood-vessels. The rupture of these vessels gives rise to repeated hemorrhages, which in turn organize and increase the thickness of the membrane. The brain may be compressed.

Symptoms.—Often no symptoms are presented. Headache, slight difficulty in memory or in thinking may be present for years. With the recurrent hemorrhages the patient may have repeated attacks of hemiplegia or monoplegia. The recurrence and rapid disappearance of apoplectic attacks are suggestive. With extensive hemorrhage convulsions or coma may be produced.

Treatment must be directed to the cause, if ascertainable.

ACUTE LEPTOMENINGITIS
(Acute Meningitis)

Definition.—An acute inflammation of the pia-arachnoid of the brain and spinal cord. The inflammation is, in rare instances, limited to the cerebral or spinal membrane, but in the great majority of cases the entire membrane is involved.

Etiology.—Acute meningitis is regularly of bacterial origin. The bacteria capable of causing meningitis are of many varieties, the diplococcus intracellularis, the tubercle bacillus, the pneumococcus, streptococci, staphylococci, the typhoid and influenza bacilli, the spirochete of syphilis. Certain of the infecting organisms cause a meningitis so distinctive in its symptomatology and course as to merit description separately. The diplococcus intracellularis is the cause of epidemic cerebrospinal meningitis. The tubercle bacillus gives rise to a distinctive clinical condition. Syphilitic meningitis requires separate consideration.

The meningitis excited by the pneumococcus, streptococci or staphylococci, the influenza or typhoid bacilli may be considered together as a suppurative meningitis.

1. CEREBROSPINAL MENINGITIS
(Cerebrospinal Fever. Spotted Fever)

Definition.—A specific infectious disease, caused by the meningococcus, occurring sporadically and in epidemics, and marked by inflammation of the meninges of the brain and cord and by severe nervous symptoms.

Etiology.—A few sporadic cases are met with every year in almost all of the larger cities. In addition, epidemics, sometimes of considerable extent, are not infrequent both in country and town. These occur almost always in the late winter and spring. Overcrowding, filth, poor food and fatigue are predisposing causes, and yet the disease appears at times among the children farthest removed from these conditions. Children and young adults are chiefly attacked.

The direct cause is a specific micro-organism—the DIPLOCOCCUS INTRACELLULARIS MENINGITIDIS of Weichselbaum — which is found abundantly in the inflammatory exudate and especially in the bodies of the pus cells. In size and shape it much resembles the gonococcus.

This germ has been found in the nasal secretion, and it is probable that infection of the meninges occurs through the ethmoidal cells. The disease is directly contagious, but susceptibility varies and communication can rarely be established.

Morbid Anatomy.—There is an acute exudative inflammation of the pia-arachnoid of the brain and spinal cord, the exudate consisting of serum, fibrin and pus in varying proportions. In the rapidly fatal cases an intense hyperemia may be the only visible sign of inflammation. Usually, however, turbid serum or pus may be seen abundantly over the vertex and base of the brain, especially in the sulci. The exudate is usually very abundant over the posterior surface of the dorsal and lumbar cord. The inflammation also involves the superficial layers of the brain and cord, and regularly extends to the sheaths of the cranial and spinal nerves. The ventricles of the brain are often distended with turbid fluid. In some of the chronic cases the meninges become opaque and thickened. There are no characteristic lesions of the other viscera.

2. TUBERCULOUS MENINGITIS, ACUTE HYDROCEPHALUS

Tuberculous meningitis is always secondary to tuberculosis elsewhere in the body, and is usually only a part of a general miliary tuberculosis in which symptoms referable to other organs are overshadowed by those of the meninges and brain. The disease is much more common in children than in adults. Foci of tuberculosis in the bronchial or mesenteric lymph-nodes, bones, joints, lungs, etc., are the sources of general infection, but it frequently happens that the primary focus is not demonstrable during life. Sometimes a fall or an injury may seem to be the exciting cause.

Morbid Anatomy.—The inflammation is confined chiefly to the pia mater of the base of the brain (hence the name basilar meningitis), and consists not only of an eruption of tubercles but of more or less fibrino-purulent exudate as well. The tubercles are most abundant in the interpeduncular space and in the Sylvian fissures, and are apt to lie in close relation to the arteries. Often they are so small as to be seen only with difficulty; sometimes they extend for some distance down the membrane of the cord and over the lateral surfaces of the hemispheres. The lateral ventricles are often distended with fluid (acute hydrocephalus) and the convolutions correspondingly flattened. The inflammation frequently extends from the pia to the subjacent cerebral cortex.

3. ACUTE SUPPURATIVE MENINGITIS

Etiology.—Acute suppurative meningitis is nearly always a secondary process. In rare cases the primary process is so slight, a bronchitis for example, that the meningitis appears to be primary. Three chief groups of cases are recognized. (1) Meningitis secondary to local infections, such as otitis media or mastoid disease, suppuration of

the nasal or ethmoidal sinuses or the orbit, infected fractures of the skull, especially fractures involving the middle ear or the nasal passages, abscess of the brain, bed sores or other suppurative processes in proximity to the spinal canal. (2) Meningitis secondary to systemic infections, such as pneumonia, infective endocarditis, septicemia or pyemia, typhoid fever or influenza, and various other acute infective diseases, such as measles, small-pox, rheumatic fever, actinomycosis and anthrax. (3) A terminal meningitis occurring as a rare complication of chronic nephritis, arteriosclerosis, gout and the wasting diseases of children.

Of these three types the first is by all means most important because it is to some extent preventable or remediable by proper treatment of the primary condition.

Morbid Anatomy.—The pia mater is congested and presents a more or less extensive exudate of fibrin, serum, and leukocytes. The exudate is most abundant along the vessels and in the sulci. It infiltrates the membrane, making it opaque and giving it a yellowish or greenish yellow color. In many cases the exudate is most abundant at the base of the brain, over the pons and the adjacent structures; in other cases the vertex is covered and the base free. If very extensive, almost the entire surface may be covered. The cerebrospinal fluid becomes more abundant than normal and turbid or purulent by admixture with the inflammatory exudate. The pia mater of the cord regularly shows the same conditions as that of the brain.

In meningitis resulting from extension from a local focus such as otitis media, the adjacent pia mater shows the most extensive changes, and the meningitis may be closely limited.

Cerebral Changes in Meningitis.—In any meningitis the underlying brain shows some inflammatory or degenerative changes. Congestion, edema and degeneration of the nerve-cells are common; an exudate of leukocytes may be found especially along the vessels.

The symptoms of meningitis are conveniently grouped into three stages, although these are by no means always well defined. In tubercular cases, there is, beside, a prodromal period of some days or even weeks during which the child feels out of sorts without being distinctly ill. The disease begins usually with headache, vomiting and fever as the chief symptoms. Sometimes it may be ushered in by convulsions. The headache is persistent and very severe; the vomiting is without apparent cause; the fever is usually not high (100° to 103° F.). There are great irritability and restlessness, and sometimes active delirium. The neck becomes rigid and the head retracted; the pupils are contracted; there may be twitching or rigidity of the extremities. Even when unconscious the child frequently utters periodically a short, hoarse cry —the so-called '' hydrocephalic cry.'' The neck is rigid; Kernig's sign is present, i.e., with the thigh flexed at 90° on the pelvis the leg cannot be completely extended; Babinski's reflex, hyperextension of the great toe, when the inner border of the foot is gently scratched, and the phe-

nomena shown in Figs. 106 and 107 may be demonstrated. This initial period is called the STAGE OF IRRITATION or excitement, and lasts only a few days.

The second stage, that of DEPRESSION, is marked by a gradual relief from the intolerable headache, and a change from irritability and rest-lessness to drowsiness and stupor. The irregular temperature con-tinues; the pulse is often slow or irregular; there is obstinate constipa-tion and the belly is usually retracted and boat-shaped. Local paralyses of the eye, face or extremities may occur, or there may be muscular twitchings or rigidity. Kernig's sign is present. Vasomotor disturb-ance is shown by the frequent presence of the so-called *tache-cérébrale* —a distinct white streak bounded by a reddish flush, which appears where the finger-nail is drawn over the skin. A moderate grade of optic neuritis is common. In the terminal stage of PARALYSIS stupor merges into deep coma; the sphincters are relaxed; the reflexes abolished; the pupils widely dilated, and the pulse grows very rapid and feeble. There
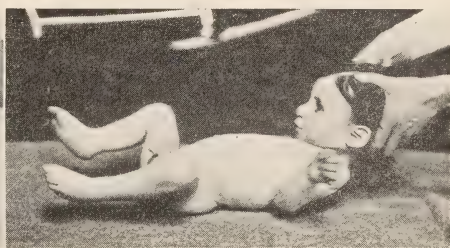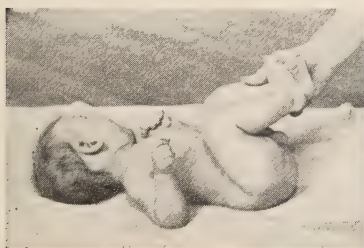
FIG. 106.—Contra-lateral reflex in meningitis. Flexion of one leg causes like flexion of the other.
FIG. 107.—Identical reflex in meningitis. Flexion of the neck causes flexion of the arms and legs. (Brudzinski.)

may be local paralyses, hemiplegia or general muscular relaxation; the temperature falls to subnormal or rises rapidly, and the patient dies in convulsions or in deep coma. The active stage of the disease commonly lasts from two to four weeks. It may, however, be fatal within a week or may extend over two or three months.

Recovery is rare, and only a very few well authenticated cases are recorded.

SPECIAL FEATURES OF CEREBROSPINAL MENINGITIS.—**Symptoms.** —The incubation period is probably less than one week. The onset is usually sudden and violent. The symptoms presented are those enumer-ated under meningitis with few important differences. In the average case the fever is higher and the course more acute than in tubercular meningitis. Certain rashes appear in the epidemic type. (a) Herpes labialis is very common. (b) A petechial rash appears in some cases, and more frequently in some epidemics than in others, upon the ex-tremities, trunk and possibly the face (spotted fever). The individual spots are of varying size and shape, but usually small. (See Fig. 108.) (c) Blebs, resembling pemphigus, occur in rare instances, and ulceration

or even gangrene of the skin may develop. At least 50%, and under serum treatment, a still higher percentage of the patients recover.

An acute inflammation of the joints occurs in about 10% of the cases.

Unusual Types of Cerebrospinal Meningitis.—The malignant form is marked by a violent onset, wild delirium, a very feeble pulse, either slow or rapid, irregular breathing, coma and death in from twelve hours to three days.

In the mild form mental symptoms are usually lacking, the course is short and the symptoms not well marked.

Abortive cases occur in which after a violent onset the fever and other symptoms rapidly subside, and convalescence begins within two or three days.

In the intermittent or relapsing form febrile intervals alternate in a very irregular way with afebrile periods of several days' duration, during which all the symptoms abate. These periods of apparent recovery are very deceptive.

Fig. 108.—The eruption of cerebrospinal meningitis. Scattered.

The chronic form is met with chiefly in children. The disease lasts many weeks or months. The child lies in stupor, emaciation becomes extreme, and bed sores and contractions of the limbs are common.

Complications and Sequelæ.—Complications such as pneumonia, pericarditis, pleurisy and nephritis are occasionally seen. Severe conjunctivitis and iritis are seen not infrequently. Sequelæ are frequent and important, and include blindness, deafness, paralyses, mental weakness, aphasia, etc.

Prognosis.—The disease is, as a rule, very fatal, but the mortality varies greatly in the different epidemics, and may range anywhere between 30 and 75 per cent.

Diagnosis.—Fever, with severe headache, insomnia, delirium and cerebral vomiting, should always suggest acute meningitis. The presence of rigidity of the neck and Kernig's sign in any case render the diagnosis highly probable. In employing the latter sign it is, however, important to know that in many persons, with the thigh flexed

to a right angle, complete extension of the leg is impossible without some force and resulting pain. In a true Kernig's sign complete extension of the leg is practically impossible.

Every added sign of meningitis, the hyperesthesia, pupillary or ocular signs, rigidity and paralysis, adds to the certainty of diagnosis.

In certain acute infectious diseases, however, such as pneumonia, typhoid fever, and the enteric infections of children, a condition (meningism) arises, which can with difficulty be distinguished from acute meningitis. This possibility must therefore be borne in mind. The symptoms of this condition are not progressive; on the contrary, they vary from day to day and may promptly clear up.

THE VARIETY OF MENINGITIS.—**Cerebrospinal.**—The presence of an epidemic, sudden onset, violent course (fever 104° to 106° F.), characteristic eruptions indicate the specific type.

Tubercular. Previous tubercular affection of bone (joints), glands, or lungs, a prodromal period of irritability and change of character, an insidious onset and rather subacute course with low fever—100° to 103° F.—suggest tuberculosis. A positive von Pirquet test is valuable in children.

Suppurative. Knowledge of an adequate cause, a local or systemic infection is here of greatest moment.

LUMBAR PUNCTURE, as a rule, renders decisive information as to the presence and the variety of meningitis.

Excess of fluid under pressure, its physical characters, and the finding of bacteria in it are the important observations.

The normal pressure in the spinal canal amounts to 100 to 150 mm. of water, and the spinal fluid drips slowly from the puncture needle. In meningitis the pressure will often rise over 200 mm., and the fluid flows rapidly or even spurts from the needle. Tubercular meningitis usually gives a perfectly clear fluid; cerebrospinal a clear or moderately turbid fluid; suppurative meningitis a turbid or purulent fluid.

If the cells in the fluid be counted, a lymphocytosis is commonly found in tubercular meningitis, a polynuclear excess in the other forms, but many departures from this rule are observed.

In the clear fluid of tubercular meningitis a fine coagulum forms in from 12 to 24 hours. On staining tubercle bacilli are found. In cerebrospinal meningitis the diplococcus intracellularis can be demonstrated, in suppurative meningitis, the pneumococcus, streptococci, staphylococci, or other causative bacteria.

THE BLOOD in meningitis shows a leukocytosis, even in many of the tubercular cases. Still, absence of leukocytosis or leukopenia suggests a tubercular infection, while very high counts (over 30,000) occur only in cerebrospinal fever. A differential count of leukocytes may show a lymphocytosis in the tubercular type; in the other forms the polynuclears prevail.

Lumbar puncture is performed with the patient lying on one side, the legs drawn as far up as possible and the head bent forward, to in-

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crease the separation between the vertebræ. The patient should be held by an assistant or secured in this position by a sheet passed under the knees and about the neck. Courageous adults may be allowed to sit on a chair or stool, with the back bent forward. An aspirating needle, 8 to 10 cm. long and 1½ mm. caliber, may be used, or, better, the Quincke needles with a relatively blunt point. The needle is inserted between the third and fourth lumbar vertebræ (the level of the crests of the ilia), either in the mid line or at one side, and directed toward the spinal canal. The dura is pierced at a depth of from 2 to 7 cm. The needle should be passed well in and then withdrawn a little.

Prognosis.—Tubercular and suppurative meningitis are practically always fatal. Recovery may occur, but is too rare to be counted upon. The mortality of epidemic cerebrospinal meningitis varies in epidemics from 30% to 75%. Under treatment by the Flexner-Jobling serum, the mortality has in some series fallen to 10% to 15%.

Treatment of Acute Meningitis.—A quiet, darkened room is required. In the stage of irritation the patient should be disturbed for examination or treatment only when necessary. The nutrition should be preserved by careful feeding, by gavage if the patient will not swallow. Careful nursing with special attention to the bed and skin is necessary to prevent bed sores. An ice-cap is to be kept continuously on the head.

Sedatives. Bromides, chloral or morphine may be required for the relief of pain in the early stages. Hot baths or packs sometimes give relief from restlessness and pain.

Lumbar puncture repeated at intervals of several days, according to the activity of the disease, as much fluid as flows freely being withdrawn each time, mitigates the symptoms.

SPECIFIC SERUM FOR CEREBROSPINAL MENINGITIS.—The anti-meningococcus serum of Flexner and Jobling has proven its value. After the withdrawal of 30 c.c. or more of the spinal fluid from 30 to 45 c.c. of the serum are injected through the puncture needle into the subdural space. In the severe cases or those coming late under treatment the larger dose is used. The injections are to be repeated at 24-hour intervals, as long as the specific diplococci can be found in the exudate, at least four doses being given. The injections should be repeated in relapsing cases.

4. SYPHILITIC MENINGITIS

Syphilitic meningitis is one of the grave results of specific infection. It is a slowly progressive process developing in the later stages of the infection. The clinical picture is not that of an acute meningitis, but of brain tumor or paretic dementia. So that in either of these conditions syphilis must be excluded by the history, the examination of the patient for active lesions or scars, the Wassermann reaction, and finally by the therapeutic test of active specific treatment.

THROMBOSIS OF CEREBRAL SINUSES AND VEINS

Thrombosis in the cerebral sinuses may occur in chlorosis and various wasting diseases without definite cause. In most cases it results from phlebitis caused by extension of inflammation from some adjacent part into the vein. Thus thrombosis of the lateral sinus follows mastoid disease, that of the longitudinal sinus follows pachymeningitis, while disease of the nasal passages or orbit may cause thrombosis of the cavernous sinus. Such thromboses are usually infected and symptoms of pyemia result.

Longitudinal Sinus.—The thrombus may be discovered only at autopsy, having given no symptoms. Venous stasis of the scalp, orbit and eye, nose-bleed and choked disk may result.

Lateral Sinus.—Thrombosis here is most often a complication of middle ear and mastoid disease. The constitutional symptoms of pyemia with chills and rapid oscillations of temperature are present. The local signs of swelling and tenderness over the mastoid, swelling and tenderness of the jugular vein in the neck, emptiness of the jugular vein below the thrombosis, and nystagmus may be present.

Aphasia may be caused and there may be severe headache, vomiting and stupor, suggesting abscess of the brain.

The sinus should be exposed through the mastoid, and after ligation of the vein in the neck, opened and emptied of its thrombus.

Cavernous Sinus.—Following disease of the nasal passages or orbit, edema of the orbit and conjunctiva with exophthalmos develops. The retina may be intensely engorged. The ocular muscles may be paralyzed and strabismus result. Suppurative panophthalmitis may follow. Symptoms of septicemia or pyemia accompany the process.

Treatment must be directed to the primary condition.

CEREBRAL ANEMIA

Anemia of the brain is ordinarily part of a general anemia due to hemorrhage, chlorosis, pernicious anemia and the like. Headache, faintness, vertigo, loss of consciousness, or even convulsions may be produced by sudden severe anemia, and similar symptoms may develop in the chronic conditions, but it is remarkable how few symptoms, except unusual fatigue, the brain commonly gives in severe anemias.

Local anemia may be produced by disease of individual vessels, or by the pressure of tumors or the accumulation of exudates (abscesses, etc.). If functionally active areas are involved, symptoms of impairment (aphasia, paralysis) may follow, but in most cases pressure rather than anemia explains these results.

CEREBRAL HYPEREMIA

Active or arterial hyperemia accompanies all the acute inflammatory affections of the brain, or may result from increase in blood pressure from any cause. Passive hyperemia results from any obstruc-

tion or impairment of the return flow, such as occurs from cardiac dilatation, emphysema, asthma, or the pressure of tumors.

There is no uniformity in the symptoms produced by hyperemia of the brain. Headache, restlessness, insomnia, delirium, mental dulness and coma have been attributed to this condition. It is difficult to separate the symptoms due to cerebral hyperemia from those produced by attendant conditions.

CEREBRAL EDEMA

Edema of the brain may accompany passive *hyperemia* and be due to the same cause. It may be part of a general *anasarca*, especially that of chronic Bright's disease. It occurs in many *local lesions* of the brain, such as atrophy of the convolutions, thrombosis of the sinuses, tumors, or abscesses.

In chronic alcoholism, a marked edema of the brain may develop, giving rise to the so-called wet brain. As in hyperemia it is difficult to separate the symptoms of edema of the brain from those of the underlying condition. (See Alcoholism, page 298.)

CEREBRAL HEMORRHAGE, EMBOLISM AND THROMBOSIS

Etiology of Hemorrhage.—Two factors are usually concerned: (1) An arteriosclerosis affecting the cerebral vessels and producing miliary aneurisms weakening their walls. Such a process may occur independently of general arteriosclerosis. (2) An increase in pressure in the vessels.

The arteriosclerosis may depend upon any of the usual factors (see page 229). Emphasis should be placed on age, for cerebral apoplexy rarely occurs before 40, and increases in frequency with advancing years. During parturition hemorrhage may be caused in the fetal head.

High pressure may be caused by the same factors as the arteriosclerosis. Chronic Bright's disease is of especial importance, causing both arteriosclerosis and high blood pressure.

Sudden increase in blood pressure due to anger, excitement, severe physical exertion, involving holding the breath and straining, the paroxysms of whooping-cough, or straining at stool may be the final determining cause of hemorrhage.

Morbid Anatomy of Hemorrhage.—Hemorrhage may occur from any vessel, but is commonly from some branch of the middle cerebral artery. Hemorrhage may be minute or may destroy most of a hemisphere. More or less extensive laceration of the brain occurs, and following this damage and the pressure of the hemorrhage, more or less extensive softening and necrosis of the brain substance. The necrosed tissue becomes yellowish or brownish and diffluent. Hemorrhages not infrequently break into the ventricles, filling them, and the blood may extend down the cord and be obtained by lumbar puncture.

Etiology of Thrombosis.—In thrombosis also there are two factors:

(1) Disease of the vessel wall—arteriosclerosis.

(2) Some changes in the blood favoring clotting, but of unknown nature. Whether thrombosis can occur from blood changes alone is an open question. Changes in the rate of flow may play a part, but these are ordinarily so slight as to render their influence doubtful.

Thrombosis may be a complication or sequel of any of the acute infectious diseases—especially syphilis. In rare instances it occurs in youth, but is common from the twentieth to the fortieth year.

Etiology of Embolism.—The usual origin of emboli is the heart, especially the lesions of the mitral valve, but emboli may come from the right side of the heart or any of the veins of the body, and passing through the wide pulmonary capillaries lodge in one of the cerebral vessels. Embolism may therefore occur at any period of life.

Morbid Anatomy of Thrombosis and Embolism.—In either condition a clot, more or less extensive, is found in some vessel, usually a branch of the middle cerebral artery. In thrombosis there is local disease of the artery wall—showing itself by atheroma, constriction, or dilatation. In embolism the wall may be normal, though, of course, an embolus may lodge in a diseased vessel, if such be present. The first effect of an embolus or thrombus is to cut off the blood supply from the territory of the plugged vessel. An area of low pressure results into which blood must be crowded from the neighboring vessels, which in the cortex intercommunicate freely. Gradually, however, circulation is restored in the periphery of the area. The central part of it, being permanently shut off from the circulation, undergoes softening and necrosis. Such necrosis is likely to be extensive at the base of the brain, in the basal ganglia and internal capsule, where the arteries are terminal. Large areas of necrosis produce cysts full of bloody fluid; the smaller give rise to congested, soft, diffluent areas of less extent.

Occurrence of Apoplexy.—Deaths from apoplexy constitute a considerable proportion of mortality statistics, about 1 in 22 of all deaths. Apoplexy is uncommon in childhood, common after 40 years, and increasingly frequent thereafter.

Symptoms. PREMONITORY.—These depend upon preceding arteriosclerosis, and include loss of mental activity, giddiness, insomnia, temporary aphasia or paralysis and the like. They are often entirely wanting.

APOPLECTIC ATTACK.—Headache, vertigo, tingling or numbness in some part of the body initiate the disturbance. In at least one-half the cases stupor appears and increases to complete coma. With the coma the face is flushed, the respiration is rapid, deep, snoring, the pulse slow and full or rapid and weak. The tension is usually high, between 200 and 300 mm., but may not be raised. The pupils are contracted or dilated, and often unequal. Conjugate deviation of the eyes is frequent, and the head may be turned toward the side of the lesion. The

temperature is normal or subnormal at first, but rises to 101° to 103° F. in many cases, and may continue for some days (resorption of blood). All the muscles are relaxed, the reflexes usually abolished, and the sphincters incontinent.

The characteristic paralysis now appears. Usually the whole of one side is involved. The cheek is puffed out in respiration, the arm and leg are flaccid, so that, if lifted, they fall like a dead weight. Unless the coma is profound, pricking the sound hand or foot will lead to vigorous motion on the affected side no response is obtained. In some cases the affected side is rigid rather than flaccid. The paralysis may be limited to one arm or leg or one side of the face.

Coma lasts a varying period and may persist till death, a week or more in some cases. Usually the coma passes off in a few hours (four). The deep reflexes return at the same time. The paralysis can then be better made out. On the paralyzed side the forehead cannot be wrinkled, the eye cannot be tightly closed, the face is flat from obliteration of the naso-labial fold, the tongue protrudes toward the paralyzed side, neither hand nor foot can be raised or moved. Lesser degrees of paralysis both in extent and degree are of course seen. The palsy may be limited to one limb or to a single group of muscles.

Course of the Affection.—In the worst cases the coma deepens, the disturbance of respiration and circulation becomes more profound, the respiration becoming irregular or Cheyne-Stokes, the pulse weak, irregular and intermittent, the blood pressure higher, and death ensues in the course of a few hours or after a week. In such cases fever is present and often mounts steadily toward the end, reaching in some instances 107° to 108° F.

Recovery occurs in most cases. After return of consciousness the paralysis begins to show improvement. This may be almost immediate or may show itself only after some days. Return of power is first seen in the muscles about the eye, so that it is often said that upper branches of the facial nerve are not involved in cerebral paralysis. Power returns in the leg before the hand and in the shoulder, elbow, hip and knee, before wrist or ankle, although the first evidence of returning power is often seen in ability to move the fingers or toes. Recovery may be rapid and complete, so that in a few days or weeks power may be restored. Usually it is a matter of weeks or months, and some paralysis, commonly of the hand and arm or the face, is left. Ability to walk is nearly always recovered. The gait is characteristic on account of the inability to raise the toe; the foot must be swung outward and the toe dragged, and the sole brought down with a flap (drop-foot). If the paralysis persists rigidity develops in the paralyzed muscles and contractures, especially in the arm and hand. Later tremor and choreiform or athetoid movements may develop. The reflexes on the paralyzed side become much exaggerated and continue so. The muscles may undergo some atrophy.

Influence of Site of Lesion.—The description given applies to

lesions involving the motor tract, the usual site, but hemorrhage, embolism or thrombosis may involve other areas and give rise to special symptoms, combined with or independent of the hemiplegia.

Aphasia is quite regularly produced by a lesion involving the left motor area in right-handed people or the right in the left-handed. It is usually recovered from, but with some permanent defect. Mental symptoms, confusion, inability to think or to remember, with lack of control and emotional excitement, may accompany hemiplegia and persist for some time. Hemianopsia of the bilateral homonymous type, that is involving the like-named (right or left) halves of both visual fields, results from lesions affecting the occipital lobes.

Word-blindness may follow lesions of these lobes. Hemianesthesia may accompany hemiplegia, but is usually only partial and quickly disappears. Cerebellar symptoms, vertigo, staggering gait and double vision may follow a hemiplegic attack. In cortical lesions paralysis may be very limited. In lesions of the internal capsule paralysis is extensive.

Lesions of one crus cause loss of ability to move the eye of the paralyzed side upward, downward or inward, the pupil is dilated and there is ptosis (third nerve). Lesions of the pons, if in its lower half, may cause paralysis of one side of the face and the opposite side of the body-crossed paralysis. In this condition the upper branch of the facial nerve is involved and the eye cannot be closed.

Diagnosis.—The focal symptoms, hemiplegia, etc., are of prime importance. If the patient is conscious, he will call attention to the loss of power, which in such cases may be limited to one extremity, or to the face. The absence of any local signs, pains or tenderness of nerve-trunks, the normal electrical reactions, as well as the sudden onset, exclude neuritis, and indicate a paralysis of central origin.

Whether such paralysis is due to definite organic lesion, such as hemorrhage, embolism or thrombosis, or is caused by the so-called functional disturbances, spasm of the cerebral vessels, or localized edema or anemia of the brain cannot be at once determined. The functional palsy will disappear in a few hours; an apoplectic lesion will cause paralysis lasting several days.

Apoplexy with coma is distinguished by the depth of the coma (the patient cannot be roused), the paralysis, the flaccidity or rigidity of the affected side, inequality of the pupils, and absence of reflexes.

Opium poisoning, alcoholism, uremic coma must be excluded. The pin point pupils and slow respiration mark opium or morphine poisoning. Alcoholic coma is less profound; the patient can be roused by pressing on the supraorbital nerves or by the policeman's method of beating the soles of the shoes with his night-stick; the speech is thick and incoherent; there is no paralysis; the pulse is rapid, full and bounding, the breath is strongly alcoholic and the stomach contents consist of food and alcoholic beverages. Uremic coma is more difficult to distinguish, especially as it may be accompanied by temporary paraly-

sis. Edema, anemia, the urinous odor of the breath, the history, if obtainable, may point to nephritis. The urine will usually be scanty, contain much albumin, many casts, and possibly blood. There is no inequality of the pupils, no deviation of the eyes, and paralysis, if present, is limited and partial.

DIFFERENTIATION BETWEEN HEMORRHAGE, EMBOLISM AND THROMBOSIS.

EMBOLISM occurs at any age, the onset is sudden without coma, the paralysis usually extensive at first clears rapidly, the pupils are unaffected, the pulse that of heart disease, the respiration normal. The presence of a definite valvular lesion, especially of the mitral, is the most convincing point.

Between hemorrhage and thrombosis, distinction is difficult and at best uncertain.

THROMBOSIS, however, belongs particularly to the syphilitic, with preceding symptoms of cerebral arteriosclerosis. Apoplexy in men below the age of 40 is very likely to have this origin. The onset is slower than in hemorrhage. The coma is not so deep, the pupils are unaffected, the pulse does not become slow, and the respiration is not so much affected (there is no increase of intracranial pressure). The local symptoms are not so extensive as in hemorrhage, and improve considerably in the first few days.

Prognosis.—Apoplexy is always grave. Complete recovery from a first attack is possible, but recurrence is almost sure to follow and attacks may be repeated for years. The patient may, however, enjoy comparative comfort in the intervals. The older the patient, the more extensive the paralysis, the deeper the coma, the graver the prognosis becomes.

Treatment.—The patient is put to bed, the head elevated. If the blood pressure is high, the face congested, the pulse strong, venesection is indicated. From 12 to 16 ounces of blood may be drawn. Nitroglycerine may be used for the same purpose, 1/100 gr. every hour or half hour till the tension is relieved. The bowels may be freely moved by two drops of Croton oil mixed with butter and dropped far back on the tongue. Absolute quiet and careful nursing are imperative. Care should be taken to secure unembarrassed breathing and to prevent the inhalation of food materials or the secretions of the mouth and throat. No effort to feed need, as a rule, be made till the patient can swallow, but food or water may be given by gavage, if necessary.

For retention of urine catheterization may be required.

In convalescence passive motion of the affected parts, with rubbing and manipulation, should be employed to maintain the muscular tone and prevent contractures. Electricity may be used for the same purpose. The patient is to be encouraged to exercise every muscle as he can.

In embolism or thrombosis, if the diagnosis is sure, the head should be kept low and stimulants administered, such as nitroglycerine, caffeine

or strychnine. If the diagnosis is uncertain these measures should not be ventured. The general measures applied to hemorrhage should then be followed.

CEREBRAL PARALYSIS OF CHILDHOOD

(Infantile Hemiplegia or Diplegia. Infantile Paralysis)

Definition.—Loss of either motion or sensation, or both, due to brain lesion. In children these conditions are frequently accompanied by defective mentality, many of the patients being imbeciles or idiots.

Etiology.—Many of these cases are traumatic, the results of cerebral hemorrhage at birth, due to protracted labor or instrumental interference. Some of them depend on an arrested development of the brain caused by a preceding lesion of some of the cerebral vessels, embolism, rupture, or thrombosis. Some are due to inherited syphilis. Not infrequently no satisfactory causation can be given.

Pathology.—The brain lesions underlying these paralyses are varied, including: 1. *Atrophy* of some greater or less part of the brain cortex due to preceding vascular lesion. The convolutions are small, the affected part, usually that supplied by the middle cerebral artery, is shrunken. Over the atrophic area there is a collection of fluid or the overlying membranes are thickened.

2. *Porencephalus,* a condition marked by the formation of holes or cavities in the brain. If such holes or cavities contain fluid they constitute cysts.

3. *Maldevelopment.* An undeveloped condition of the brain, the cells resembling those of the new-born, without other gross lesion.

4. *Hydrocephalus*—either external or internal. (a) External hydrocephalus, the collection of excessive fluid on the surface of the brain, a very rare condition. It doubtless results from meningitis or atrophy, the fluid in the latter case being poured out to fill the empty space. (b) Internal hydrocephalus, an unusual accumulation of fluid in the ventricles due to obstruction in the iter or the fourth ventricle (foramen Magendie), preventing the normal flow of fluid from the lateral ventricles to the subdural space. With hydrocephalus the ventricles are distended, the cortex thinned, until only a thin sac remains. In hydrocephalus the head is large; it may be enormous. The fontanelles are abnormally long in closing, or remain open. The measurements are increased in all diameters. In the other conditions described the head is small (microcephalus) and often asymmetrical.

Symptoms.—Onset. These patients are rarely seen at the onset of the primary trouble, hemorrhage, embolism or thrombosis, which most often underlies the permanent affection, but are brought to the physician for the paralysis or mental condition. If the condition precedes birth, the onset is not observed and only the sequelæ or permanent conditions are noted. At birth cerebral hemorrhage causes difficulty in making the child breathe, or cry, cyanosis, possibly convulsions, and often death.

After birth, paralysis most often follows one of the acute infectious diseases, measles, scarlet fever, influenza, etc., and is ushered in by convulsions, fever, vomiting and stupor. The paralysis is noticed at once and runs the course of apoplexy in later life. Later symptoms or sequelæ include: (1) Motor defects, (2) sensory defects, (3) mental defects.

(1) Motor defects. Usually on one side, sometimes on both, we find the conditions characteristic of hemiplegia, loss of power, spasticity, contractures and exaggerated reflexes. The loss of power is partial only. The patient can walk, but with the characteristic gait; and may be able to use the hand, but awkwardly. Spasticity is most marked in the legs, and if both are affected (paraplegia) the knees overlap in walking. Contractures are most marked in the upper extremity, both elbow and wrist being flexed and incapable of extension. Rarely there is no spasticity, and no increase in reflexes.

(2) Sensory defects include blindness, hemianopsia, deafness, rarely loss of smell or taste.

(3) The mental defects are of all grades. The idiots are incapable of grasping new ideas and cannot be taught to keep themselves clean. The imbecile can be taught cleanliness and grasps simple ideas, may learn to recognize persons and to speak a few words, but is far below normal standards of mentality.

Associated disturbances. (1) In the paralytic limbs athetoid or choreiform movements may occur. The latter simulates the quick, jerky movements characteristic of chorea. Athetoid movements are slower, more rhythmical, flexions and extensions, or rotations of joints, such as those of the fingers or wrist. These may occur only upon attempts to use the limb or may be constant. (2) Epilepsy is a common accompaniment; the attacks may be general, or of the Jacksonian type. (3) Aphasia is common in those suffering from right hemiplegia.

Prognosis.—This is always unfavorable. The lesions and the resultant failure of brain development are permanent.

Treatment.—For apoplexy in a child the same general measures as apply in adults are to be used. For the after-results, the paralysis and mental defects, in the severer grades nothing can be done. In the milder grades special education in institutions or under trained teachers may bring improvement. Massage and passive motions of the paralyzed limbs may be helpful.

Double spastic paraplegia (Little's disease) has in some cases been benefited by cutting the posterior nerve-roots belonging to the affected parts.

Operations upon the heads of these children for the relief of an assumed pressure due to microcephaly or for the drainage of cysts have proven fruitless.

HYDROCEPHALUS.—If this precedes birth the large head may be an obstacle to delivery, sometimes requiring the sacrifice of the child. Often the child appears normal at birth; the enlargement of the head

and failure of the child to develop first attracting attention about the sixth month. The size of the head and the failure to develop mentally are first noted. The fontanelles remain open and fluctuate. The size of the head increases steadily. Nystagmus, strabismus and blindness from optic atrophy are common. Muscular weakness is marked, so that the child cannot sit or stand, and may not be able to raise the head. A spastic condition in all four extremities with atrophy from disuse is common. Vomiting is frequent. Convulsions occur from time to time. In these severe cases the child never learns to walk or talk and usually dies within the first year or two. In the milder grades the child grows and develops mentally. A slight hydrocephalus is not inconsistent with full mental ability. Between these limits there are many cases in which great enlargement of the head is associated with defective power in one or both sides of the body and imperfect mental development.

ABSCESS OF THE BRAIN

Etiology.—Abscess of the brain results from the lodgment of pyogenic micro-organisms. These secure admission either (1) *directly* from some local focus—otitis media, either acute or chronic, suppuration of frontal or ethmoidal sinuses or of the orbit, from penetrating and infected wounds of the skull or operations—or (2) *indirectly* through the blood stream, in pyemia, malignant endocarditis, septicemia, pneumonia and the like conditions in which pyogenic cocci are found in the blood. The second group is of lesser importance because the conditions are practically always fatal. In the first group of cases there is hope of prevention or adequate treatment.

Morbid Anatomy.—The abscesses may be single or multiple. The pyemic abscesses are usually multiple. The single abscess most often results from local infection. The abscess is large or small, and may occur in any part, but is most frequent in the temporo-sphenoidal lobe, because of its relation to the ear. The pus is usually greenish and foul. Any of the pyogenic cocci may be found in it. A suppurative meningitis, either local or general, commonly accompanies the abscess.

Symptoms.—These are notoriously vague and unsatisfactory. The rational symptoms of suppuration, chills, fever, sweating, anemia and emaciation, may be present, but are rarely observed unless from the primary process. Fever is ordinarily slight, may show remarkable fluctuations, is often absent. A leukocytosis is quite constant, and the differential count may show an increase of the polynuclear cells.

The cerebral symptoms are those of increasing intracranial pressure and an abscess in this respect closely resembles a tumor of the brain. Headache is common, severe and persistent. Vomiting may occur quite regularly. The pulse is often slow, respiration slow and irregular. The pupils may be dilated or unequal. Optic neuritis (choked disc) may develop, but is often wanting. The mental state is one of indifference or stupor; convulsions may occur.

Focal symptoms. Muscular rigidity or paralysis, aphasia and sen-

sory disturbances, if the proper areas of the brain are involved, may be present, but are often lacking, especially in abscesses secondary to otitis media.

Ocular symptoms, such as inequality of the pupils, conjugate deviation or strabismus, or paralysis of one or more muscles, may be observed.

The COURSE of abscess of the brain is as variable as its symptomatology. The cases may be grouped as follows: (1) Latent. The abscess for months or even years gives few symptoms, a little headache, an occasional rise of temperature, mild prostration, and is not recognized till a terminal suppurative meningitis or the autopsy reveals the condition. (2) Tumor-like. The symptoms are slowly evolved and suggest tumor of the brain. (3) Fulminant. The course may be very short and sharp, the symptoms closely reproducing the picture of acute suppurative meningitis.

The termination is usually marked by the extension of the inflammation to the pia mater of the brain and cord, a suppurative meningitis.

Diagnosis.—The most important point is the cause. Obscure or severe cerebral symptoms following otitis media, acute or chronic, a fracture of the skull, etc., should always cause suspicion of the presence of an abscess.

Signs of cerebral irritation, persistent headache, vomiting, stupor, convulsions or coma, without meningitis—slight rigidity of the neck, no Kernig's sign, no Babinski reflex, clear cerebrospinal fluid, especially if accompanied by fever, and leukocytosis are important.

Focal symptoms, paralysis, aphasia, deafness on one side, hemianopsia, are only rarely present. Tenderness of the skull to pressure or percussion immediately over the abscess is unusual. The localization must be made in most cases from the cause. If from ear disease, the abscess is looked for in the temporo-sphenoidal lobe; if from a nasal condition, in the frontal lobe; if from a fracture, in relation to the bone lesion.

Prognosis.—The outlook is always grave. Formerly always fatal, about 50 per cent. of the patients are now saved by proper surgical treatment.

Treatment.—Prophylaxis is most important. The aseptic or antiseptic management of fractures of the skull, suppurations of the ear, and other possible sources of cerebral abscess is essential. When an abscess has formed, trephining and drainage are essential.

GENERAL PARESIS

(Dementia Paralytica.   Progressive Paralysis of the Insane)

Definition.—A chronic progressive disease of the brain and cord, marked by motor and psychical disturbances and ending in complete paralysis and dementia.

Etiology.—General paresis is a disease of adult life, affecting men much more often than women, due to the strain of modern life, and,

in most cases, the remote effects of syphilis.   Syphilis is held to be the basis of the disease, and for this reason the disease frequently presents itself in both husband and wife.   From five to twenty-five years may intervene between the initial lesion and the development of symptoms of paresis.   Excess of any kind, hard work, worry, over-indulgence in alcohol, are secondary factors in causing the disease.   General paralysis is very closely related to locomotor ataxia.

Morbid Anatomy.—Changes are found both in brain and cord.   In the brain PACHYMENINGITIS, with thickening and cloudiness of the pia mater and adhesions between the two membranes, is common.   With these changes ATROPHY of the cerebral cortex, particularly the frontal and parietal lobes, shown by thinning of the convolutions and deepening of the gyri and diminished weight of the brain, is combined.

ARTERIOSCLEROTIC CHANGES in the cerebral vessels constitute the third important feature.   HYDROCEPHALUS, both internal and external, is common.

Microscopically the changes in the vessels and inflammatory infiltration of the pia and dura are associated with degenerative changes in the cortex, especially of the ganglion cells.   Many of these disappear, those remaining show marked degeneration.   The neuroglia fibers, the spindle cells and nuclei of the neuroglia are increased.

The changes of general paresis are not limited strictly to the frontal and parietal regions, but are found to some extent in other parts, even in the cerebellum and pons.

Symptoms.—The onset of paretic dementia is peculiarly insidious and gradual.   The symptoms are both mental and physical.   Often the initial symptoms are neurasthenia, mental depression, headache, sleeplessness, dizziness, fatigue.

Defects of judgment then appear.   A man becomes careless in his habits, neglectful of his person, of his business, of his relations to his family and others.   Petty thefts may be committed, checks signed heedlessly, those about him subjected to evidences of irritability or anger without reason.   In some cases an exaggerated love of showy dress, or extravagance in expenditure, or excessive sexual indulgence marks the loss of self-control.   Defects of memory become marked.   The patient's age, or address, or the names of well-known persons or places are forgotten.   He becomes unable to spell or to do simple sums in arithmetic. Things brought to his attention are immediately forgotten, until the mind becomes a complete blank.   Delusions of grandeur are not uncommon, the patient imagining himself to have become enormously rich, or powerful, or to be engaged in large undertakings.   Melancholic or depressive delusions occur in some cases, but are unusual.   In all cases there is a progressive apathy or indifference to surroundings and to his own mental state.   Convulsions or apoplectic attacks are common.   The convulsions may be general or of the localized Jacksonian type.   The attacks of paralysis are transitory, either hemiplegia or aphasia.   In fatal cases no sufficient cause for the paralysis can be found.

The PHYSICAL SIGNS are striking. (1) Eye phenomena. Reflex immobility of the pupil. Reaction to light alone may be lost (Argyll-Robertson pupil) or to both light and accommodation. The pupils may be unequal, or irregular, or paralyses of ocular muscles may develop. (2) Facial signs. (a) Disturbances of speech are among the earliest and most important symptoms. The patients have difficulty in pronouncing consonants, particularly l and r, and the defective memory shows itself strikingly in inability to repeat simple series of words joined by sound rather than by sense, such as "truly rural," "third artillery brigade," or "round the rough and ragged rock the ragged rascal ran." (b) Tremors of the facial muscles and the tongue are commonly present. (3) Ataxia. Muscular movements of any kind are awkward and associated with tremor. The writing shows this particularly well. The letters show the tremor of the hand, the words are written in any direction and letters or syllables are omitted. (4) The deep reflexes are lost or exaggerated, especially the Achilles and patellar reflexes. (5) Muscular weakness is pronounced, especially late in the disease, when the patients are bedridden; in consequence atrophy results from disuse. (6) Trophic disturbances occur late in the disease. Bed sores, perforating ulcers of the foot, or gangrene of an extremity may develop. (7) The blood gives a Wassermann reaction in 75% to 80% of cases. The cerebrospinal fluid regularly shows an unusually large number of lymphocytes.

Course.—The disease is progressive and ends in dementia. The duration is usually several years—from 2 to 15 or 20. In the end the patient becomes bed-ridden, devoid of intelligence, and loses power over the sphincters, so that the urine is retained or passed involuntarily, and the feces are passed unconsciously. He dies of exhaustion or of intercurrent disease, such as cystitis or septicemia.

Prognosis.—Recovery is possible but exceedingly rare. Remissions may occur, lasting for years.

Diagnosis.—Typical cases can hardly be mistaken. In the early stages the disease may be confused with neurasthenia. The combination of mental symptoms with the definite physical signs of paresis should distinguish it. The Wassermann reaction and examination of the cerebrospinal fluid are helpful. Disseminated sclerosis, especially in its terminal states, may be confused with paresis. The mental condition should be sufficient to make the distinction. Cerebral syphilis is distinguished with difficulty, because the diseases are closely related and cerebral syphilis may terminate in general paresis. The course of the disease under treatment is the surest guide. Cerebral syphilis will improve more promptly and decisively than will paresis.

Alcoholic dementia is marked by a history of alcoholism, the physical evidences of chronic alcoholism, possibly peripheral neuritis, an absence of the pupillary signs, and by characteristic hallucinations and delusions.

Treatment.—Vigorous antisyphilitic treatment, salvarsan followed

by mercury by injection or inunction and increasing doses of potassium iodide, up to 30 or 60 grains thrice daily, should be tried in specific cases. The Wassermann reaction may be followed, but should not control the treatment.

Complete mental rest, careful feeding and judicious supervision, such as can ordinarily be obtained only in a sanatorium, are indicated in all cases. The bromides or chloral may be used for epileptic seizures or convulsions. Hydrotherapy and massage are helpful in the early stages.

MULTIPLE SCLEROSIS
(Disseminated Sclerosis; Insular Sclerosis)

Definition.—A chronic affection of the brain and cord, due to replacement of scattered areas of nerve-cells or nerve-fibers by fibrous tissue.

Sclerosis of the central nervous system occurs under a great variety of conditions, in which the fibrous tissue appears as a replacement process, designed to fill the gap left by destruction or degeneration of the nerve tissue, or as a terminal step in an inflammatory process. Sclerosis regularly includes thickening of the pia arachnoid and increase in the neuroglia tissue of the cerebrospinal axis. Thus it is seen in more or less extensive areas of the brain following vascular lesions, as in the cerebral palsies of childhood or in tabes dorsalis, lateral sclerosis, the ascending and descending degenerations, following transverse lesions of the cord, or following meningitis or other inflammatory lesions.

But independently of all such conditions sclerosis occurs in isolated or disseminated areas of the brain and cord as an independent affection.

Etiology.—The disease occurs frequently at all ages in either sex. Many causative factors have been advanced, such as syphilis, or other acute infectious disease, exposure to cold and wet, and the like; none of them is sufficiently constant to be important.

Morbid Anatomy.—The essential feature is areas of fibrosis of the brain and cord scattered at haphazard throughout them. The brain or cord may show areas of atrophy corresponding to the sclerosis. Opaque patches in the pia mater may overlie the placques of fibrous tissue, and the affected areas may be firmer than normal. On section these areas are found to be gray or grayish red foci of connective tissue. Microscopically an increase of fibrous tissue with disappearance of nerve-cells and nerve-fibers in the affected part of the cord or brain is found.

Symptoms.—As these must depend upon the location and order of development of the sclerotic foci in the brain and cord the clinical picture necessarily varies greatly, the characteristic feature being that the symptoms present evidences of involvement of various areas of the brain and cord independent both in time and position of one another. In most cases the following symptoms are noted:

1. Spinal. (a) Paraplegia, usually of spastic type. More or less difficulty in walking develops, possibly with spasticity, and overlapping of the knees as the feet are advanced. (b) Intention tremor, usually

most marked in the hands, but also in the legs, or in neck and face. The tremor is rapid, from 4 to 7 per second, and varies in degree, so that it may be slight or so severe as to render such acts as drinking water almost impossible. This tremor appears only on effort; at rest the muscles are quiet. Charcot's explanation that the tremor results from difficulty in the transmission of motor impulses through sclerosed areas seems to best fit the case. (c) Difficulties in micturition and defecation may appear early, and are regularly present late. Delay in starting the urine, retention, or incontinence may develop. Constipation is the rule; fecal incontinence develops in the final stages. (d) Sensory disturbances, tingling, numbness, shooting pains, formication and the like may affect either upper or lower extremities. (e) Reflexes. The tendon reflexes are exaggerated. The abdominal skin reflex is absent. Babinski's reflex is present.

2. CEREBRAL. (a) Scanning speech. The speech is slow and measured, the voice monotonous. In the later stages each word or even syllable must be pronounced slowly and with effort. The difficulty in speech may be associated with tremor of the facial muscles. (b) Ocular symptoms. Diplopia from partial or complete paralysis of ocular muscles, nystagmus on movement of the eyes, or even at rest, or inequalities of the pupils may be present. The *nystagmus* is very characteristic. Blindness may result from optic atrophy. (c) Vertigo or vomiting may occur. (d) Mental debility, shown by impairment of memory, false hopefulness or undue depression, or lack of emotional control, may be shown.

Course and Prognosis.—The course of the disease varies remarkably. Remissions lasting years occur and symptoms come and go. Even optic atrophy has been recovered from. As a rule the disease is progressive and incurable, yet the patients live for years.

Diagnosis.—From hysteria, multiple sclerosis is distinguished by the ocular symptoms, especially nystagmus or optic atrophy, the intention tremor, disturbances of the bladder, the Babinski reflex. General paresis presents earlier mental disturbances with a history of syphilis, the Argyll-Robertson pupil, diminished or lost knee-jerks, the presence of a lymphocytosis of the spinal fluid, without nystagmus, intention tremor or the scanning speech. Paralysis agitans is marked by tremor which is constant, not intentional, and without nystagmus, scanning speech, exaggerated reflexes, or Babinski's sign.

Treatment.—Life in a mild climate, freedom from labor or anxiety, a nutritious diet and exercise carefully regulated to prevent fatigue are to be enjoined. Medicines cannot affect the underlying process. Constipation and other symptoms are to be treated as they arise.

TUMORS AND CYSTS OF THE BRAIN

Etiology.—Brain tumors occur at all ages, in men more often than women. A preceding tuberculous or syphilitic affection may be of import, otherwise the etiology is not known.

Morbid Anatomy.—Many varieties of brain tumor are known—the common ones are tubercle, sarcoma, glioma, gumma and carcinoma. Tubercle occurs in the form of large, isolated and usually single tumors made up of masses of miliary tubercles or a single large tubercle as much as an inch or an inch and a half in diameter. Tubercle in the form of tumor is common in childhood, rare in later life. Gumma, on the other hand, never results from inherited syphilis, and is therefore very rare in childhood, but common in adult life. Sarcoma or carcinoma may follow a primary growth elsewhere, but is usually primary in the brain. Glioma is a growth of neuroglia, and therefore peculiar to the nervous system. Cystic tumors of the brain may arise from parasitic infections, such as echinococcus or cysticercus. They are very uncommon in this country. Fibroma, angioma, myxoma, osteoma and other rare forms are known.

In brain tumor the intracranial pressure is always increased, and if the skull is opened during life the dura bulges and its pulsation is diminished. The cerebral convolutions are regularly flattened. The ventricles often contain excess of fluid owing to obstruction of the venæ Galeni by pressure.

Symptoms.—These are both general and focal.

GENERAL.—These are produced by the steadily increasing intracranial pressure. (1) Severe and persistent headache. (2) Mental disturbance. The disposition may change notably, and the patient become irritable, careless or indifferent. Memory is impaired and attention fails. The patient may become dirty and untidy in his habits. (3) Vertigo and vomiting. Dizziness is common in any cerebral tumor, but is pronounced if the cerebellum is involved. Vomiting is frequent, and is usually projectile in type. (4) Slow pulse. The rate is slow, 50 to 60, the pulse full and of high tension. (5) Convulsions. These may be general or of the Jacksonian type. (6) Choked disk is present in 80 to 90% of brain tumors, and constitutes one of the most important signs of brain tumor. Amblyopia, defective vision or blindness may accompany the choked disk. The field of vision may be contracted with inversion of the color-fields, so that the field for blue may interlace with or lie within that for red.

FOCAL SYMPTOMS.—These depend upon the location.

Frontal Region.—Changes in temperament and mental activity are notable. Memory is impaired, ability to concentrate thought or to learn diminishes, and the patient becomes stupid and irritable, or uncontrollable.

Tumors of the third left frontal convolution in right-handed persons cause motor aphasia with agraphia. In left-handed persons the tumor must be in the right hemisphere.

Motor Area.—Tonic or clonic spasms of the muscles of some portion of the body are caused by tumors of the motor cortex. The spasms so caused occur at intervals and may terminate in general convulsions. The spasms always begin in those muscles whose centers of

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representation are most affected by the tumor. Therefore, observation of these spasms as to their location at the beginning and the mode of spreading helps greatly in locating a tumor. Either extremity or the face may be affected. Paralysis or paresis may follow either in arm, leg or face, but cortical tumors rarely produce hemiplegia. Tumors involving the internal capsule or the motor tract beyond it may cause hemiplegia.

Parietal Region.—Disturbances are not constant, but in many cases impairment of sensation or muscular sense follows. Word-blindness has been found to be due to lesion of the inferior parietal lobule (left).

Occipital Region.—Hemianopsia (bilateral homonymous) results from lesion of the occipital lobe. It may be unknown to the patient and must be tested for.

Island of Reil.—Tumors of this area produce paraphasia—that is, disturbance of speech in which one word is regularly substituted for another. They may produce pressure upon the neighboring face centers or even upon the internal capsule.

Crus.—Tumor involving the crus should produce paralysis of the third nerve of the same side and the extremities of the opposite side of the body. The fourth nerve may also be involved.

Pons.—Tumors in the upper part involve the third and fifth, in the lower part, the fifth, sixth, seventh and eighth. In this situation paralysis of one or more of these nerves with loss of power on the opposite side of the body, so-called alternating hemiplegia, may result.

Medulla.—The ninth, tenth, eleventh and twelfth nerves suffer with resulting difficulties in swallowing, in respiration, irregularity of the pulse, and paralysis of the tongue. Polyuria or glycosuria may also result.

Cerebellum.—Because of the small space in which the cerebellum lies enclosed by the tentorium cerebelli, small tumors produce severe results. By compressing the fourth ventricle, tumors in this region cause distention of the ventricles above, and hence symptoms appear early. Staggering gait, vertigo and cerebellar ataxia, an incoördination which entirely disappears when the patient lies down, are marked. Nystagmus is frequently present. Knee-jerks are exaggerated.

Diagnosis.—The symptoms of steadily increasing intracranial pressure with the presence of choked disk are characteristic. Abscess of the brain must be excluded by the absence of any cause for suppuration, absence of any constant leukocytosis, or fever, or other constitutional symptoms of suppuration and the presence of optic neuritis. Brain abscess rarely gives rise to definite focal symptoms and then only late in the disease.

Tubercular meningitis causes a more rapid development of symptoms, with hyperesthesia to light or sound, possibly tubercles in the chorioid, and without optic neuritis. Tubercle bacilli should be found in the spinal fluid. Chronic hydrocephalus in children is excluded by the size and shape of the head, the symmetry of the rigidity or loss of power, absence of choked disk, and the comparative comfort of the patients.

The Wassermann reaction is a valuable aid in the diagnosis of gumma.

Course and Prognosis.—Gradual but steady increase in symptoms over one or two years is the usual history. Death at the end of two or three years results in nearly all cases. Gummata may be relieved, possibly cured by specific treatment. A few tumors have been successfully removed by surgery, but only ten per cent. are operable, and of those operated upon but few survive.

Treatment.—Active antisyphilitic treatment should be tried. The iodide of potassium is given in increasing doses up to 60 grains thrice daily. Inunctions of mercurial ointment or injections of mercury may be combined with the iodide. Careful study should be made in the effort to locate the tumor and operation undertaken, if it is accessible.

APHASIA

Definition.—Literally the word means loss of the power of speech, but in medicine includes all the disturbances of the use of language, either written or spoken, not dependent on intellectual failure or paralysis of the vocal organs. Aphasia is a symptom of various cerebral lesions, not a disease in itself. The exact determination of the defect in speech is of material aid in the location of cerebral lesions, especially brain tumors. The subject is, therefore, of sufficient importance to be dealt with separately.

To understand the different types of aphasia one must go back to the mental processes underlying the use of words in speaking or writing. Every word has four points of relation or centers in the cerebral cortex. It can be heard or seen, it may be spoken or written. Thus the auditory center lies in the temporal lobe, the visual center in the angular gyrus, the motor speech center in the third left frontal convolution, and the center for writing in close relation to it in the centers for arm and hand. Each of these actions depends upon the integrity of a definite area of the cortex, and also the normal use of words in speaking and writing depends not only upon integrity of these centers, but on their free communication with one another through the association fibers of the brain.

The chief types of aphasia are described thus:

1. SENSORY APHASIA.—1. *Word-Deafness.*—This condition is best illustrated by the common experience of forgetting names. Every one has at some time seen an acquaintance whom he sees, recognizes, perhaps knows all about, and yet cannot name. The memory picture of his name cannot be recalled and hence cannot be expressed. But if now the acquaintance says my name is John King, the recollection may still fail, if the defect is complete, but usually the sound of the name at once recalls its word picture and one recognizes its propriety and utters it. The condition here described is, of course, a matter of momentary forgetfulness, but in true word-deafness the condition persists. The patient is therefore unable to name familiar objects when

brought to mind in any manner. In this condition there must be a cortical lesion involving the sound memory centers in the temporo-sphenoidal lobe. If the defect is but partial, the name of a thing cannot be recalled of one's self, but is recognized at once when heard. This auditory amnesia, as it is sometimes called, implies the integrity of the word-centers, but a defect, sub-cortical, in the fibers connecting them with other centers. This distinction between cortical and sub-cortical lesions is of importance mainly with reference to the possibility of operating upon cerebral lesions such as tumors.

2. *Word-Blindness.*—If this condition is complete word pictures are entirely lost. The thing seen is not recognized, and hence cannot be named. If the defect is partial only, the missing word picture, not reached by one route, the eye for example, can at once be recalled if the approach is made through another channel such as the ear. Thus a patient with complete word-blindness cannot recognize and name a bell, for example, when seen, but if it is rung in his ear he recognizes it at once and on request draws it in outline. He has the concept of a bell with its form, but it cannot be reached through the visual route.

With relation to language complete word-blindness is shown by inability to read or write. The patient cannot read because he cannot recognize the characters before him. He cannot write because he cannot recall the shapes of the necessary letters. If the defect be only partial, he can write at dictation or copy, although not understanding what is written.

Complete word-blindness indicates a cortical lesion in the region of the inferior parietal convolutions and angular gyrus. A partial word-blindness is produced by a sub-cortical lesion involving the fibers of association.

3. *Intercortical Sensory Aphasia.*—The fibers of association between the auditory and visual word centers may be interrupted, producing a condition in which an object seen cannot be named, or if named cannot be recalled to mind, although recognized in both instances. This condition is produced by lesions of the association fibers connecting the temporal and occipital lobes.

2. Motor Aphasia.—1. *Aphemia*, or what is commonly called simply aphasia, a condition in which although words or things are recognized by eye and ear, the patient cannot name them or speak because of inability to recall the muscular movement necessary. This happens, of course, independent of paralysis of the muscles of speech. 2. *Agraphia*, the condition in which one can name objects and speak as usual, but cannot write, because the memory of the movements necessary is lost. 3. *Intercortical motor aphasia or paraphasia.* In this condition an interruption or interference with the association fibers connecting the motor and other word-memory centers results in a disturbance characterized by the use of the wrong word in speech or writing. The patient speaks or writes jargon. Paraphasia is particularly associated with lesions of the island of Reil.

PLATE VII.

The Fundus in Amaurotic Family Idiocy.

AMAUROTIC FAMILY IDIOCY

Definition.—A rare affection of Hebrew children, characterized by mental impairment during the period of infancy, blindness and loss of power over much of the body.

Etiology.—The causation is unknown. The disease occurs only among the Hebrews, and often affects more than one child in a family. It is not syphilitic.

Morbid Anatomy.—A degenerative process is found affecting all the nerve-cells of the brain and cord. The degeneration affects particularly the body of the cell and the dendrites, not the axis cylinder. The changes in the cell may be slight or so complete that the cell is disintegrated. Sachs thinks the disease due to congenital deficiency of the nervous system, such that after three or four months of normal life, the cells yield to the ordinary demands of life and disintegrate.

Symptoms.—These are briefly summed up by Sachs as: (1) Mental impairment during the first few months of life leading to complete idiocy. (2) Paresis or paralysis, either flaccid or spastic, of the greater part of the body. (3) The reflexes may be deficient, normal or increased. (4) A diminution of vision terminating in absolute blindness. (5) Marasmus and death before the age of two years.

Various other symptoms, such as increased sensibility to touch or sound, nystagmus, strabismus, or convulsions, have been noted in some cases.

Diagnosis.—This rests upon the mental impairment, the blindness and the ophthalmoscopic examination, which shows, in the region of the macula lutea, a bright cherry-red spot, an absolutely pathognomonic sign. (See Plate VII.) Treatment is of no avail.

FUNCTIONAL NERVOUS DISEASES

ACUTE DELIRIUM

(Typhomania. Brain Fever. Bell's Mania)

Definition.—An acute delirium or mania accompanied by fever, regularly fatal, without adequate organic lesions.

Etiology.—Emotional excitement, injury, toxemia and infection are given as causes. In most instances no satisfactory explanation is to be had.

Morbid Anatomy.—The meninges are congested, but the brain appears normal. Microscopically Osler found some exudation of leukocytes about the vessels and in the lymph-spaces.

Symptoms.—The onset may be preceded by several days of restlessness, irritability and insomnia. The onset is usually marked by fever, 102° to 104° F., with some increase in the pulse and prostration. Wild delirium or an active mania develops easily. Hallucinations are vivid, the patient talks or sings incessantly, and keeps in constant motion.

Such acts as salaaming may be repeated endlessly. The patient may struggle with his attendants, but rarely injures anyone but himself.

A typhoid-like condition is developed with fever, a dry-brown tongue, rapid pulse, marked prostration, and rapid emaciation. At the end of a week or ten days the patient dies in collapse.

Diagnosis.—Typhoid fever must be excluded by absence of the characteristic roseola and the enlargement of the spleen, and by the study of the leukocyte count, the Widal reaction, and blood cultures. Pneumonia can be excluded only by the absence of the usual symptoms and repeated careful physical examinations. Delirium tremens presents a distinct etiology, characteristic hallucinations, and the active tremor of tongue and hands.

Acute meningitis may be excluded by the absence of rigidity of the neck and Kernig's sign, and by the results of lumbar puncture. It is evident that the diagnosis of acute delirium can hardly be safely ventured before the autopsy reveals the absence of lesions sufficient to account for the disease.

Treatment.—Blood-letting has been recommended in the early stages. The general treatment must be that of typhoid fever. The bromides gr. xxx t. i. d. may be given for excitement or hyoscin in doses of 1/100 gr. Dram doses of the fluid extract of ergot every two hours are recommended.

PARALYSIS AGITANS

Definition.—A chronic nervous affection marked by muscular weakness, tremors and rigidity.

Etiology.—Two-thirds of the patients are men. The disease rarely begins before forty. Exposure, privation and nervous strain appear to favor its development. Numbers of cases were observed during the siege of Paris and the war of the rebellion.

Morbid Anatomy.—No distinctive lesions have been found. Brawny patches in the skin have suggested myxedema, and the theory that lesions of the parathyroids were related to the disease has been advanced.

Symptoms.—The onset is either sudden or gradual. Usually the weakness, stiffness and tremor appear in one hand or arm, then the other. All four extremities and even the head may be affected. When fully developed the clinical picture includes:

1. The tremor, the most striking symptom. It is a rapid tremor, four to eight oscillations per second, usually most marked in the hand. The movement of the fingers suggests pill-rolling. Flexion and extension of the wrist or slight rotation may be combined with the finger movements. It may show clearly in the hand-writing. It usually is present at rest, and disappears on effort, but in some cases accompanies voluntary action—*i.e.,* is *intentional.* In the foot the tremor causes rapid flexion and extension at the ankle, and the toe taps the floor as in clonus.

2. Rigidity of the muscles is the most pronounced feature of the disease. Rigidity precedes or accompanies the tremor. Passive motion may be impeded, but the reflexes are not increased and ankle clonus is not found. The facies becomes mask-like. The eyes are held open and fixed in one position, the whole head being revolved instead of moving the eyes from side to side. The patient rarely winks. The neck muscles are rigid as in torticollis, but both sides are involved, and the face is not turned to either side. The body is bent forward at the hips and the spine is bowed forward. In the hands similarly the fingers and wrists are usually flexed. Speech and gait are often characteristic. In speaking the patient hesitates a moment, then hurries the words out and stops abruptly. In like manner the patient, rising from a chair, halts a moment, then with the body bent forward advances with short, hurried steps, as though the feet were trying to overtake the body. In rare cases the patient actually falls forward.

3. The mental state is marked by inertia. The patients are dull, sluggish, talk little and are indifferent. Dementia is often suggested, but when roused the patients show complete possession of their faculties.

4. Sensory disturbances. The patients complain of sensations of heat or cold, but respond normally to tests. Vasomotor changes may be indicated by flushing of the skin, increased sweating or salivation.

5. Muscular atrophy is not marked and no electrical changes are present.

Course.—The disease is chronic and incurable, lasting many years. Periods of improvement may occur.

Diagnosis.—In a typical case the diagnosis can be made on sight. The attitude of the body, the gait, the mask-like face, and muscular rigidity as well as the tremor are characteristic. Disseminated sclerosis is marked by earlier onset, scanning speech, and nystagmus. The tremor is regularly intentional. Post-hemiplegic tremor can be excluded by the history of preceding paralysis and the increased reflexes. Hysteria must be considered.

Treatment.—Medication appears to be of no avail. Massage, electricity and vibration have been of service. The administration of extracts of the parathyroid glands has been advised.

ACUTE CHOREA

(Chorea Minor. Sydenham's Chorea. St. Vitus' Dance)

Definition.—A nervous affection, common in children, characterized by irregular involuntary muscular contractions, resulting in purposeless movements, and often accompanied by psychic disturbances.

Etiology.—The disease is common between the ages of five and fifteen, especially in girls. A neurotic heredity can often be traced. The strain of school-life is usually the chief factor. Sudden fright or ex-

citement often precedes the appearance of symptoms. About 25% of the patients give a history of antecedent rheumatism. Any acute infectious disease or other condition lowering the vitality of a child may be an accessory cause of chorea.

Morbid Anatomy.—Death from chorea is rare and pathological studies relatively few. Minute emboli in the cerebral vessels have been found in some cases, and have led to the theory that chorea is caused by numbers of such emboli taking their origin from a rheumatic endocarditis. This theory covers but a part of the cases.

Perivascular hemorrhages and areas of infiltration in the brain have also been observed. The pathology is by no means established.

Bacteria.—Many investigators have found bacteria in the brain, but the organisms have been of wide variety, usually streptococci. Poynton and Paine found their streptococcus rheumaticus in the cerebral embolisms. The dependence of the disease upon bacterial infection is not established, but the theory that it is due to the action of toxins, probably bacterial, upon a sensitive nervous system receives much support.

Symptoms.—Motor.—Irregular, jerky, purposeless movements of one or both upper extremities are first noted. The movements may be confined to one extremity or extend to all. The face may be affected. With these movements awkwardness and weakness in the use of the hands or other parts develop. The patient drops things because of the chorea and is often rebuked or punished for carelessness. Speech may become hesitating, jerky and imperfect. The movements may be very slight, occurring at rare intervals and for some time scarcely attracting attention. In the mild form they cease during sleep. They are regularly increased by excitement or embarrassment, and are therefore often most noticeable during examination. In severe cases the jerky movements affect all parts of the body, are almost constant, more violent, and may persist during sleep. The weakness may develop into paralysis.

Psychic.—Irritability, peevishness and inability to concentrate the attention attend the milder cases. In severer forms the mental disturbance becomes more marked. Delirium with varied hallucinations may develop. Stupor and dementia have been observed in fatal cases. Constitutional anemia, malnutrition and loss of appetite are regularly present. The pulse is rapid and the patients feeble. Fever appears only in the severer cases.

Complications.—Heart murmurs are common. They may be hemic or due to definite endocarditis. The location of the apex impulse, the size of the heart, the character of its action, and the circulatory conditions must all be considered in attempting to decide this question. Acute pericarditis occasionally develops. Other rheumatic complications, such as erythema nodosum, purpura and subcutaneous nodules, may be seen.

Course.—The disease runs its course in two or three weeks or

months. It regularly ends in recovery. Occasional choreic movements may be seen for months afterward. Death is possible in the severe forms.

Treatment.—Freedom from disturbing influences of any kind, fresh air and good food are the prime requirements. The home conditions must be made favorable or the child removed from them. Relief from school duties, an out-door life, and nutritious food will promptly relieve mild cases. In pronounced cases rest in bed is necessary.

Arsenic is regularly administered. Fowler's solution in doses of 2 to 5 minims, thrice daily, is commonly given. Some increase the dose to the limit of intolerance, but this seems undesirable.

Chloral and the bromides are often given for sedative effect. Massage and passive movements may be employed. In chronic cases suggestion and educative exercises are of value.

CONVULSIONS OF CHILDREN
(Infantile Convulsions)

Definition.—General tonic and clonic convulsions occurring in childhood from various causes, not including epilepsy.

Etiology.—A family tendency to nervous disturbance is notable in some cases; in others the individual seems to have an abnormally sensitive nervous system. The origin of this weakness can often not be made out. In children of such hereditary or acquired nervous susceptibility any form of trauma, toxemia or infection may be the cause of a general convulsion. The possible causes of convulsions in childhood, therefore, include practically all the varied forms of disease or injury to which they are subject. Certain influences are so important as to be specially enumerated. 1. Gastro-intestinal disorders of any kind, especially overloading the stomach with indigestible food, and constipation, are the commonest causes of convulsions in childhood. Intestinal parasites and inflammations of any kind are important factors.

Dentition is commonly regarded as the most frequent cause of infantile convulsions. In most instances it is some associated alimentary disorder and not the "cutting of the teeth" that causes the disturbance, yet in very sensitive children "teething" alone may cause convulsions.

2. In childhood the invasion of an acute infectious disease, especially pneumonia, scarlet fever, whooping-cough or small-pox, is frequently marked by a convulsion. The initial chill commonly seen in adults at the onset of such acute infections seems to be replaced in childhood by a convulsion.

3. Rachitis predisposes children to convulsive seizures.

4. Any illness or indisposition lowering the vitality of a child favors these convulsive seizures.

5. Fright or excitement may precipitate a convulsion in a susceptible child.

Symptoms.—The convulsions may occur in a child apparently in perfect health or they may be preceded by irritability and restlessness with evidences of digestive disturbance.

The seizures vary from attacks of brief unconsciousness with little or no spasm to general tonic and clonic convulsions not to be distinguished from the characteristic fits of epilepsy.

In the milder attacks, the so-called "inward spasm," the child becomes silent, rigid, stares or rolls the eyes upward, the face or one hand twitches for a moment and the paroxysm is over.

The severer paroxysms begin in like manner, but the whole body becomes rigid, the hands clinched, the elbows flexed, the head retracted, and quick spasmodic jerkings of the face and extremities follow. The respiration is embarrassed, the pulse becomes rapid, feeble, and possibly imperceptible. The face and lips become deeply cyanosed and death appears imminent. There may be frothing at the mouth and rattling of mucus in the throat. After lasting a few seconds or several minutes the convulsion gradually ceases, the muscles relax, and the child passes into a stupor from which it rouses slowly, perhaps hours afterward. The attacks may be repeated at any time; frequently several follow one another at short intervals, and many may occur in a day. Death rarely follows a single convulsion, but may result if the convulsions are frequently repeated.

The convulsions are likely to recur, but may not. In most cases they cease entirely as the child grows older. Persistence of the convulsions usually means epilepsy.

Diagnosis.—The convulsion can be recognized on sight or history. The cause is the important question and must be sought with care. A thorough physical examination and a study of the diet and life of the child should be made to determine the conditions which have caused the convulsions. Epilepsy can be excluded only after prolonged observations, but it is to be remembered that convulsions are common, epilepsy in infancy relatively rare.

Treatment.—During the convulsion chloroform should be given by inhalation. If the tongue is protruded or caught between the teeth a bit of wood or rubber should be used as a gag to prevent injury to it. A hot bath or hot pack, with mustard added to the water in the proportion of a tablespoonful to the gallon, should be given at once with the object of reducing internal congestion by bringing the blood to the surface. The bowel should be emptied by an enema, and castor oil given by mouth. Chloral and the bromide of soda may then be given by rectum, 5 grains of chloral and 10 of the bromide to a child of a year or more. If the danger of recurrence seems great, morphine sulphate, gr. 1/50 for a child of one year, may be given hypodermatically.

Quiet and a restricted diet should be required for several days before allowing return to the ordinary mode of life. Careful examination should then be made for any possible cause of convulsions. The diet and regulation of the life are of prime importance. Rachitis if

present must be treated. Adenoids may require removal. Anemia should be treated, and in every way the general health of the child maintained at the highest point. Excitement or excess of any kind must be avoided. If under such care convulsions are repeated the probability of epilepsy is increased.

EPILEPSY

Definition.—A nervous affection characterized by periodic attacks of unconsciousness commonly associated with general convulsions and frequently preceded by an aura.

Etiology.—Epilepsy regularly develops in childhood in either sex, and rarely begins after the age of thirty. Heredity plays an important part. If epilepsy itself is not present in the family, neuroses of some kind are often found.

Syphilis, tuberculosis or alcoholism in the parents predisposes to epilepsy. Every influence which lowers the nervous vigor of a family favors the development of epilepsy. Similarly any influence which lowers the nervous vigor of the individual may in a susceptible person precipitate the first attack or cause the repetition of the convulsions. (a) Toxic influences, such as alcohol, tobacco, or the toxemia resulting from overburdening the alimentary tract and constipation, are of first importance. (b) Acute disease of any kind, especially the acute infectious diseases, may precede the onset. (c) Reflex irritations from eye-strain, adenoids, intestinal parasites, uterine or ovarian disease, phimosis and the like. (d) Mental shock or excitement are important agencies. (e) In females the attacks are likely to occur at the time of menstruation.

The cerebral hemorrhages of birth, injuries to the skull at that time or later, defective cerebral development from any cause, all favor epilepsy. Idiocy or imbecility and epilepsy are frequently associated.

Morbid Anatomy.—Many brain lesions have been found in patients suffering from epilepsy, especially defects of development, porencephalus, hemorrhage, cysts, tumors, meningitis and the like. These lesions are not essential, however, but may be regarded as sources of irritation which favor the development of convulsions. Recently careful studies have shown that in those dying of epilepsy degenerations in the cerebral nerve-cells, especially those of the second cortical layer, can be demonstrated. These degenerations involve especially the nucleus and nucleolus.

Epilepsy is still, however, regarded as a functional disease—*i.e.,* without definite anatomical basis.

Symptoms.—1. GRAND MAL.—The seizures of general convulsions are the typical form of the malady. These include a tonic stage, a clonic stage, and a period of stupor or coma, and in many cases are preceded by an aura.

The Aura.—A warning of some kind occurs in many cases; it may be motor, sensory or psychic. Motor. A tremor, a jerk or movement of

some part or parts of the body, rapid revolution, or running forward a few steps are sometimes seen. Sensory auræ are more common. Tingling, burning, numbness, a certain sound, a flash of light and various other unusual sensations are recorded. Psychic auræ present themselves as a certain mental depression or exaltation, certain emotions, or recollections. Whatever the aura it is repeated, as a rule, before each fit. The aura may just precede the paroxysm, but sometimes an interval of some minutes elapses before the onset. Auræ are present in a majority of patients but not in all.

Tonic Stage.—The patient suddenly becomes unconscious and falls, often injuring himself about the head and face in falling. The whole body becomes rigid. The head is drawn back or to one side, the eyes are turned upward and to one side, the face is pale, the thorax immovable (apnea), the limbs flexed. Often the sudden contraction of the respiratory muscles causes an inarticulate cry. The tonic stage lasts but a minute or two.

Clonic Stage.—The convulsive movements begin in the face or an extremity, but quickly become general. The eyes, face, limbs and whole body are thrown into violent jerky contractions, the teeth are ground together, the tongue is protruded and often bitten, the saliva is churned into froth and pours from the mouth, the respiration is jerky and imperfect, the face becomes cyanotic, the pulse rapid and weak. The contractions last from one to five minutes, and then gradually die out. Both bladder and rectum are commonly evacuated during the paroxysm. Stupor or complete coma follows, lasting a half hour or more. The patient then awakes, possibly unconscious that he has had a convulsion, but usually weak, depressed and mentally confused.

Recurrence.—Epileptic fits recur after varying intervals, hours, days, months or years. The fits often occur in groups. Fright, fatigue, excitement and the like may precipitate an attack, but usually they recur without apparent cause. In most cases the attacks slowly increase in frequency.

Nocturnal epilepsy is common, the fits coming on at any time during the night, even in sleep. Under these circumstances the patient may not know what has happened or may be aware from the muscular soreness, exhaustion or the bitten tongue that he has had a fit.

Status Epilepticus.—In some instances, especially late in the disease, the fits are repeated in a long series, are accompanied by fever, even 104° to 105° F., and followed by collapse. Such crises end favorably, as a rule, but death may occur.

Postepileptic State.—Usually the mind quickly clears, but it may remain clouded. A trance-like condition may supervene, and mania with homicidal tendencies is well known to follow in some cases.

2. PETIT MAL.—Temporary unconsciousness with few or no convulsive signs marks this condition. The manifestations are varied. In some a sudden pallor, a fixed stare, a momentary obliviousness, are all that are seen. Slight tremor of the eyes, face or an extremity may be seen.

The patient may fall or may drop what he is holding. The urine may be passed involuntarily.

In other cases the patient turns rapidly about, or runs a few steps in an automatic manner, and then returns to his usual condition.

Psychic Equivalents.—In rare cases the epileptic seizure is replaced by some unconscious and automatic act. Murder has been done, and fires set by the irresponsible victims of such seizures. In other cases long journeys have been taken or various complicated acts performed. Ordinary convulsions occurring before or after such paroxysms reveal their nature.

Course.—The disease tends to grow worse with steady increase in frequency of the convulsions and increasing mental dulness in the intervals. The patients may die from their falls or injuries received while unconscious (drowning) or from the convulsions, or in a condition of dementia.

Diagnosis.—The typical fit can hardly be mistaken. The aura, tonic and clonic spasm and subsequent stupor are very characteristic. Repetition of the convulsions is highly suggestive. The presence of scars or bruises on the scalp or the tongue is helpful. Nocturnal fits are regularly epileptic. In children under two years convulsions are often repeated a number of times and then cease, without subsequent epilepsy. The family history is important, but time is required to settle the question. In adults the convulsions of uremia are sometimes mistaken for epilepsy. The anemia, cardiac changes, arteriosclerosis and urinary findings should differentiate the condition.

Hysteria is easily recognized. The fit is overdone. The patient is noisy, never injures himself or herself, but may strike others; the convulsion is exaggerated, unconsciousness is feigned, but there is no relaxation of the sphincters and no coma.

3. JACKSONIAN EPILEPSY.—(Cortical, symptomatic or partial epilepsy.) In this condition the seizures are limited to one extremity or to one side of the face. The convulsion may extend from one part to another, as from the arm to the leg or vice versa. The localized form may at any time give place to a general convulsion. Jacksonian epilepsy usually arises from focal irritation of the brain, such as a depressed fracture, a localized meningitis, a tumor or abscess. Similar attacks may occur in uremia. The location of the focal lesion is usually indicated by the part in which the convulsion begins.

Prognosis.—Some few patients recover, but the disease is generally incurable and tends to be progressive.

Treatment.—GENERAL.—The patient must be examined thoroughly for any defect which may be a source of irritation. Eye-strain, adenoids, uterine displacement, any possible source of local irritation should be relieved. The digestion should be studied, an easily digestible diet prescribed, and constipation avoided. An out-door life with freedom from annoyance or care is helpful. Moderate exercise is desirable. Excess of any kind must be forbidden.

MEDICINAL.—Bromide of potassium or sodium is effective in reducing the frequency of the paroxysms. Small doses, 5 grains, are given, and the dose later increased. If acne appears, the medicine must be reduced or stopped. It may be continued indefinitely. Many other nerve sedatives, chloral, cannabis indica, zinc, etc., are employed.

INSTITUTIONAL.—Control of the mode of life of the epileptic patient is essential. If this cannot be secured at home, removal to a sanatorium should be urged. Great numbers of epileptic patients are now treated advantageously in farm-colonies, such as the Craig Colony, Sonyea, N. Y. Jacksonian epilepsy may be relieved by trephining and appropriate treatment of the focal lesion.

MIGRAINE
(Megrim. Sick Headache)

Definition.—An affection characterized by attacks of severe headache, often attended by disturbances of vision and digestion. Migraine presents a close analogy to an epilepsy in which the motor discharges are replaced in large part by sensory phenomena.

Etiology.—The explanation of migraine is regularly based on two factors, a constitutional nervous defect or weakness, and some local disturbance or weakness. (a) Constitutional. Heredity is regarded as important. Some neuropathic taint (headaches, epilepsy, neurasthenia and the like) can usually be found in the family. A gouty tendency is sometimes present and excess of uric acid in the blood or deficiency of it in the secretions is commonly invoked as explaining the attacks of megrim. Exact proof of the relation of either of these conditions to megrim is, however, lacking, and the common accusation of uric acid in this regard is without adequate foundation.

(b) Local conditions. These comprise most functional and organic defects, such as simple constipation, eye-strain, adenoids, nasal spurs and polypi, uterine or ovarian disease and the like. In general any influence which lowers the health of the patient or acts as a source of local irritation may have relation to attacks of migraine. In many cases it is quite impossible to determine the etiology. The affection appears in families without neuropathic taint and in healthy individuals. The attacks begin in childhood or early adult life, more often in females.

Symptoms.—Prodromal disturbances announce to some the approaching attack. The prodromata are varied, languor, drowsiness, flashes of light, spasm of ocular muscles, dilatation of the pupil and the like. The headache itself usually begins in the morning. The pain is referred to any part of the head, usually unilateral, sometimes sharply localized. Visual disturbances commonly accompany the pain. The sight is blurred, or there may be definite hemianopsia.

In some cases varied figures with flashing colors play before the eyes. Nausea and vomiting are common; the vomitus is yellow from the presence of bile. Vomiting is severe in some persons and may give relief.

Tingling and numbness may be felt in the face or arm, and may be accompanied by weakness or actual loss of power. Speech may be slow and stumbling and temporary aphasia may develop. The face is usually pale from vasomotor constriction, and later the face and ear on the affected side may flush. The patient is greatly prostrated and hypersensitive to noise or disturbance of any kind. The attack usually lasts throughout the day or several days. The patient is prostrated for some time afterward.

Course.—The attacks occur at intervals of days or weeks, sometimes from definite indiscretions in diet or life, but often without apparent reason and despite the best of treatment. They tend to become less frequent and less severe in later life. The menopause seems to bring relief to women. Many patients having sought relief in vain finally resign themselves to their suffering, and yet are able to live lives of activity and usefulness.

Treatment.—THE ATTACK.—A brisk saline cathartic taken at the first signal of the attack is advisable. Washing out the stomach with hot water is recommended in those cases where vomiting is severe. Black coffee given at the onset is sometimes helpful. During the attack the patient must be kept in bed and heat applied to the feet. Acetanilid, phenacetin and caffeine are given for relief of the pain, and are sometimes helpful, if they can be retained. Morphine may be required for the very severe attacks, but should be withheld, if possible. Often the after-effects of morphine are so disagreeable to these patients that its relief is declined. Hot or cold applications, as preferred, may be made to the head.

GENERAL.—In the intervals the life of the patient should be carefully planned. A vegetarian diet helps some, but dietary restrictions are usually valueless. Avoidance of such articles of food as cause gastric disturbance usually commends itself to the patient. Excesses of any kind should be avoided. Abundance of sleep and rest is advisable. An out-door life and moderate exercise are recommended.

LOCAL.—Every defect should, if possible, be corrected. Thus errors of refraction, adenoids, bad teeth, digestive disturbances, especially constipation, uterine or ovarian disease and the like must be appropriately treated.

NEURALGIA

Definition.—A painful affection of a nerve or nerves without discoverable lesions either in the nerve itself or adjacent tissues to explain it.

The term is a broad one which commonly is loosely used to cover all types of pain. Every effort should be made to distinguish neuralgia, on the one hand, from pain excited by definite nerve changes such as occur in neuritis, and on the other from pain due to definite local lesions, such as abscesses, new growths and the like. Even with the most careful study, however, we may be unable in some cases to distinguish neuralgia from neuritis.

Etiology.—The affection is rarely met with in childhood, but is common among adults, especially women. A neurotic tendency or heredity underlies many cases. The weak and anemic are most subject to it. Constitutional conditions producing weakness and anemia may precede it, especially infectious diseases, such as influenza and malaria, or the systemic poisons of rheumatism, gout, diabetes or chronic nephritis, or alcohol, lead or arsenic. In those liable to the affection, exposure to cold and wet, or any condition producing nervous exhaustion or debility may precipitate an attack. In not a few cases no cause can be found and we must be satisfied to call them idiopathic. The term ought not to be applied to pains which can be explained by definite pathological lesions, such as caries of the teeth, suppuration in the antrum of Highmore, pressure of tumors and the like. The more carefully patients are studied the less use we shall have for this designation.

Symptoms.—Neuralgia presents itself in the form of paroxysms of pain, usually described as sharp, burning or stabbing, in the territory of a certain nerve or nerves of one side of the head or body. The paroxysms last for a brief time or may be protracted. In the intervals there is usually no discomfort. The pain is usually severe, and may be agonizing. Regularly there are no changes seen in the affected area. There may, however, be vasomotor or finally trophic changes in persistent cases. Thus the skin may be hot and red, or cold and pale. Edema, atrophy or induration of the skin, or whitening of the hair in chronic cases may be seen. Where herpes or such changes as these are met with we are probably dealing with manifestations of definite lesions of the nervous system. (See Herpes Zoster.)

The attacks of pain are likely to recur at regular intervals of a day or days, without relation to malaria. The pain often shifts from one nerve to another. Along the course of the affected nerve there may be tender points, especially where it emerges from a canal or becomes more superficial. The affection lasts for varying periods, weeks or months, or in some cases persists for life. Any nerve in the body may be affected. The more important varieties follow:

(1) TRIGEMINAL NEURALGIA. Tic Douloureux. Any of the branches of the fifth nerve may be involved. The attacks of pain are characteristic. The affection is often severe and protracted, and may require heroic measures for relief. Before accepting the diagnosis of neuralgia we must carefully exclude organic lesions, such as the pressure of tumors, cerebral or in the course of the nerve, disease of bone causing pressure in the course of the nerve, or any cause of peripheral irritation, such as caries of the teeth, suppuration of the antrum or the frontal or ethmoidal sinuses, or the growth of tumors within the orbit, nose or mouth. In neuralgias of the ophthalmic division the possibility of glaucoma, or errors of refraction or muscular defects of the eyes must be considered.

(2) INTERCOSTAL NEURALGIA.—This is frequently a severe type of the affection. Care must be taken to exclude pressure on the nerve by diseased vertebræ (Pott's disease) or aneurisms or other tumors. Pleurisy

or other inflammatory lesions must be ruled out. The association with herpes zoster is frequently seen in this form of the disease.

(3) Lumbar Neuralgia.—Lesions of the spine, of the kidney or other abdominal viscera must be sought for, stone in the kidney especially. Herpes zoster is frequent in this type also.

(4) Neuralgia of the Upper Extremity.—Here we must look particularly for causes of pressure upon the brachial plexus or the nerves of the arm, involvement of the shoulder-joint or adjacent bursa, and remember that neuritis is much more common than neuralgia.

(5) Neuralgia of the Lower Extremity.—Sciatica is considered separately. For neuralgic pains in the feet we most often find explanation in the presence of affections of the arch (flat-foot) or disease of the bones (especially tuberculosis). The metatarsalgia of Morton is a
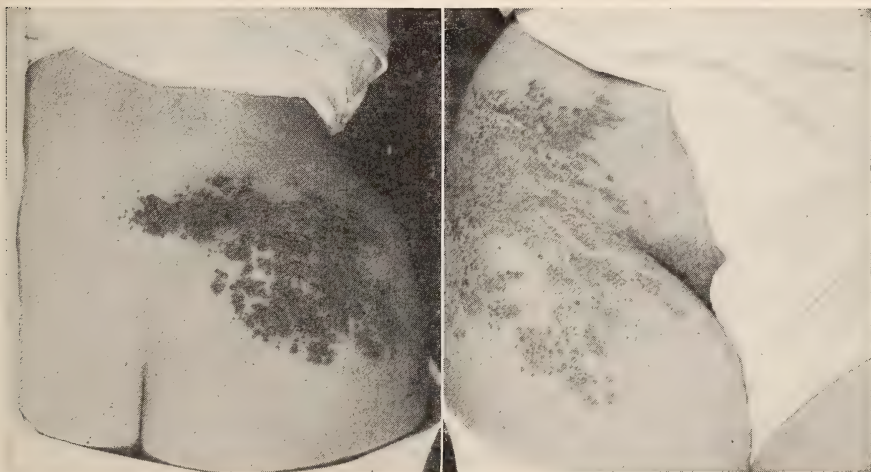
Fig. 109.—Herpes zoster, lesion of lower lumbar nerves. A, fresh lesions; B, older, drying lesions and pigmentation.

neuralgic affection usually due to pressure upon one or more of the nerves of the foot. The possibility of locomotor ataxia is to be remembered. The condition of the veins and arteries must be considered.

(6) Herpes zoster is a neuralgic affection of the intercostal nerves dependent upon an acute interstitial inflammation of the dorsal root ganglia and therefore not properly included among functional affections. Clinically it is, however, best considered in this relation. The affection is characterized by an attack of pain referred to the territory of one or two intercostal or lumbar nerves on one side of the body, associated possibly with slight fever and constitutional disturbance. The skin of the affected area is hyperalgesic. After three or four days an eruption of many fine vesicles appears along the course of the nerve, usually in several scattered groups, sometimes extensive (see Fig. 106).

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In the course of a week or ten days the vesicles dry up and disappear, leaving brownish pigmentation and in some instances minute scars. As a rule the pain lessens with the development of the eruption and ceases with its disappearance; in rare instances it persists indefinitely.

Diagnosis.—The common organic causes for pains often called neuralgia have been suggested. Neuralgia must also be distinguished from neuritis. The latter affection can usually be recognized by more general tenderness along the course of the nerves, the presence of marked trophic or vasomotor changes, loss of sensation or paralysis in the affected part or the electrical reactions of degeneration.

Prognosis.—Usually the affection is readily amenable to treatment. It may, however, prove protracted, and in the severer types, suicide is not unknown. The affection is never fatal in itself.

Treatment.—(1) Constitutional.—The adequate treatment of debility and anemia is essential. Rest, fresh air, good food and tonics, such as iron, strychnine, quinine, cod liver oil, the hypophosphites, or glycerophosphates may be employed. In the poorly-nourished forced feeding is advantageous. In severe cases a favorable climate and agreeable surroundings are helpful. The withdrawal of the use of alcohol or any other poison, such as lead or arsenic, is indicated. Influenza, malaria, rheumatism, gout or diabetes, if present, must be properly treated.

(2) Symptomatic.—The relief of the attacks of pain. (a) The common analgesics, aspirin, phenacetin, antipyrin or antifebrin may be given in appropriate doses. The possibility of harm from their use, especially if continued for any length of time, must be borne in mind. Their effects on the heart and circulation should be carefully watched. Strychnine given in full doses hypodermatically is sometimes valuable. Aconitia, in doses of 1/200 to 1/150 grain may be tried, and is a successful but also a dangerous remedy. Morphine should be used only when everything else has failed. The danger of a habit is great. (b) Local treatment. Applications of heat or cold (the hot-water bottle or ice-cap) may be of help. Various anodyne liniments are employed, such as chloroform, belladonna, chloral and camphor, menthol. The actual cautery is valuable, where it can be used, as on the trunk or extremities. The galvanic current may be of service.

Surgery is the last resort. Division of the affected nerve, or resection, or finally the excision of the ganglia, in the case of the fifth nerve, the Gasserian, may be required.

TETANY

(Tetanilla)

Definition.—A rare affection characterized by intermittent tonic cramps of the extremities, often painful, and usually without constitutional or cerebral disturbance. The condition is a symptom-complex (rather than an independent disease), associated with a great variety

of conditions varying from parathyroidectomy to intestinal infections.

Etiology.—This condition has recently been demonstrated to be due to a poverty of the blood and tissues in calcium salts caused by loss of function on the part of the parathyroid glands. The peculiar localization of the spasms is as yet unexplained.

Tetany is met with at all ages, but especially in children. It has been observed in many different conditions, but is most often seen in (a) gastro-intestinal disease in children, especially the rachitic, (b) dilatation of the stomach in adults, (c) in pregnancy, and (d) following thyroidectomy, the parathyroids being unwittingly removed with the larger gland.

The parathyroids in man vary from two to four in number, and are minute glandular bodies lying in close relation to the posterior border of the thyroid, and therefore easily removed with it.

Symptoms.—The cramps first appear in the hands, later in the feet. Escherich insists that in children the larynx is frequently involved, and instances of involvement of the face and thoracic muscles are known, but the manifestations are commonly limited to the hands and feet. The cramps are bilateral and symmetrical. In the upper extremity the wrists are flexed, the thumb is drawn into the palm, the fingers flexed at the metacarpo-phalangeal joints but otherwise straight, giving the appearance known as the accoucheur's hand. In the lower extremity the foot is partly extended, unusually arched, the toes turned inward and closely adducted. Various modifications of the attitude of the hand may be observed, especially that in which the thumb is drawn strongly into the palm and the fingers flexed upon it. The spasm is tonic, intermittent, and can be excited by various forms of irritation, mechanical or electrical. The spasm is increased by pressure upon the nerves or arteries of the affected limb (Trousseau's sign), indeed by pressure anywhere upon the limb, or even the opposite member. The electrical excitability of the muscles is increased (Erb's sign). Tapping over a nerve or upon a muscle excites spasm in the affected muscles (Chvostek's sign). Chvostek's sign is best brought out by tapping the facial nerve in front of the ear. Much has been made of these special signs, but they are not constant and are not always easily elicited. Trousseau's sign is most reliable. The sensory nerves have also been shown to be easily excited.

The spasms in the extremities come and go, enduring for a varying length of time, lessening as the primary condition improves in cases of gastro-enteritis, or persisting till death in fatal cases. The condition when seen in adults with gastrectasia is regularly fatal.

In the severe cases the spasms are evidently painful and the strength of the patient is sapped by their recurrence. The spasms may spread to the face and involve even the respiratory muscles. There is no fever or constitutional disturbance, except such as belongs to the original disease or results from exhaustion.

Diagnosis.—The spasms are easily recognized on sight. The vari-

ous signs noted distinguish them from hysterical spasms. The primary rigidity of the neck and jaw with constitutional symptoms readily distinguish true tetanus.

Treatment.—The indicated treatment in these cases at present includes the administration of preparations of the parathyroid glands and the salts of calcium. Preparations of the parathyroids are now offered by several manufacturers of glandular extracts. These preparations are not standardized and the dosage varies. Of the calcium salts the lactate is preferred. It may be given by the mouth, subcutaneously or intravenously. In a case of gastric tetany in an adult Kinnicutt gave intravenously 4 grams dissolved in 1,000 c.c. of normal salt solution daily for several successive days. An equal quantity can readily be given by mouth, when the stomach is tolerant. Hot baths may give temporary relief from the spasms.

The underlying condition, rachitis, gastro-enteritis or gastric dilatation, must receive appropriate treatment. In gastric cases operation for the relief of pyloric stenosis causing dilatation must be considered, but is very likely to prove fatal because of exhaustion.

OCCUPATION NEUROSES
(Professional Spasms)

Definition.—Irregular involuntary cramps of groups of muscles commonly employed in the repetition of certain acts peculiar to the occupation.

Etiology.—Occupations requiring the continuous repetition of more or less complicated muscular movements, such as writing, telegraphing, typewriting, piano-playing and the like, are the essential cause. The affection, therefore, appears only in persons thus occupied. Practically every occupation which requires the continual repetition of certain muscular movements has its peculiar neurosis. The general condition, especially that of the nervous system, influences the development of the condition. Overwork, worry, excitement, fatigue from any cause favors it. Tuberculosis, diabetes, alcoholism, or any constitutional disease lowering the vitality of the patient predisposes to it.

Symptoms.—These consist of cramps of the muscles or groups of muscles employed in the performance of the occupation, in most cases the muscles of the hand and arm, associated with more or less sensory disturbances. The condition is very closely akin to the cramps induced by over-fatigue.

The difficulty develops very gradually as a rule, slowly increasing for months or years, before the patient seeks advice, but it may develop suddenly. On attempting the usual movements the muscles are thrown into spasms which render their action difficult, awkward and irregular, or even impossible. More or less pain accompanies each attempt, and the subject is ultimately forced to give up his occupation. The pain is often in the shoulder or elbow, rather than the hand. A fine tremor

develops in the hand. Actual paralysis of muscles is seen only in advanced conditions.

Sensory disturbances are usually present. An unusual sense of fatigue or strain in the muscles involved is common. Anesthesia, hyperesthesia or paresthesia may be present, but are uncommon. The electrical reactions are normal. Mental anxiety and depression are natural and are commonly marked.

Course.—The affection tends to become chronic and incurable. The disability may vary with rest or improvement in the general condition.

Treatment.—The general condition of the patient must be considered, and the health improved by appropriate means. The use of alcohol and tobacco should be limited or stopped. Anemia or malnutrition should be appropriately treated.

In milder cases a change in the method of working may be helpful. Thus in writer's cramp, the free-arm movement in writing may be taught, or the left hand substituted for the right. Sometimes the use of a very large pen-holder is an advantage. Similar measures may be employed in other callings.

Massage, electricity, in various forms, hot and cold douching, or vibration may be employed locally. Active and passive exercise of the affected muscles in movements other than those in which cramp occurs is valuable. In many cases nothing but a complete change of occupation avails.

HYSTERIA

Definition.—Möbius defines it as a state in which ideas control the body and produce morbid changes in its functions.

Etiology.—As the name suggests, the disease is much more frequent in women, but is not unknown in men. Puberty and adolesence are the favoring periods for its development, but the disease may appear in childhood, or continue to old age. Hysteria occurs in all countries and among all nations, but the Latin, Slav and Israelite are especially susceptible. Heredity and education are the chief predisposing causes. The children of neuropathic parents are apt to inherit sensitive and unstable nervous systems. Faulty education often develops the emotional powers and fails to teach the necessity of self-control. The victim of such conditions under any strain readily presents the phenomena of hysteria. The immediate cause of hysterical attacks is usually some mental or physical shock, thus sudden fright, grief, worry or trauma of any kind, or the debility produced by disease may directly precede the outbreak. Imitation has at all times played an important part in producing hysterical manifestations, especially in endemic and epidemic waves. Much of the demoniacal possession and of the witchcraft of ancient days is now recognized as hysteria. Schools, hospitals and religious gatherings have always been favorite fields for this disease.

Symptoms.—These are best classified according to the French, who have devoted the most exhaustive study to the disease, into two great groups: (1) the stigmata, which are essentially persistent symptoms, and

(2) the accidents, which are transitory. The stigmata of hysteria are sensory, motor and psychic.

SENSORY STIGMATA.—(1) Anesthesia. This is most often confined to one side of the body, a hemianesthesia, but it may affect any part of the surface and the mucous membranes of the affected parts may be anesthetic. Except in the form of hemianesthesia, the hysterical affection does not correspond to the distribution of any of the peripheral nerves or to areas supplied by any segment of the cord. Thus an anesthesia appears in a single extremity, or as a band about the trunk or the limb, or in the form of a glove or stocking. The anesthetic zones are movable. Under the influence of suggestion employed by the application of metals, magnets, or the electrical current the area of anesthesia may be rapidly shifted from one part to another, or from one side of the body to the other. In some cases the elements of sensation are disassociated and the anesthesia may be confined to loss of sense of pain or touch. It is characteristic of hysterical anesthesia that the patient is usually ignorant of this loss of sensory power until it is developed by examination. The loss of sensation may be associated with loss of motive power or may be entirely independent of it. The organic and tendon reflexes are not modified by hysterical anesthesia, as in organic lesions showing lack of sensation. The pupillary, abdominal, cremasteric, knee and toe reflexes are preserved.

(2) Hyperesthesia is very common. Headache referred to the vertex or occiput is frequent. The pain is often intense, and from its being compared to the driving of a nail into the head, has earned the classical designation of clavus hystericus. Pain in the back either localized or diffuse is an almost constant feature of hysteria. The hypersensitiveness may be confined to definite areas upon the trunk, especially of the abdomen, and may therefore suggest disease of the underlying viscera, such as the stomach, the appendix or the ovaries. Hyperesthesia of the whole abdomen may be very suggestive of peritonitis. Pressure upon any hypersensitive point may promote an hysterical fit. Such points or areas are then spoken of as hysterogenic points or zones.

MOTOR STIGMATA.—Like the sensory stigmata, these are unknown to the patient, and must be elicited by examination. They differ only in degree from the motor accidents, from which, however, they should be clearly distinguished. (1) Movements in hysteria are retarded, the degree of retardation varying as do the other symptoms. (2) Coordination is imperfect and movements are consequently awkward. (3) More than one movement at a time is usually impossible. (4) Voluntary power as measured by the dynamometer is weakened. (5) There is a tendency to rigidity and contracture of various muscles.

All of these motor stigmata are usually most marked in parts affected by anesthesia. The basis of these motor phenomena is the defective cerebral control of the patient, and they may be lessened by evoking active mental effort, or intensified by suggestion.

MENTAL STIGMATA.—The mental stigmata of the hysteric may be

summarized as loss of will power and memory with an abnormal impressionability or susceptibility to suggestion.

Suggestibility.—The mind of an hysterical person is abnormally sensitive to the slighest suggestion or impression, and an idea once admitted, seems to develop to an extreme without any participation of the will or of the personal consciousness of the subject. This characteristic leads to imperfect and whimsical actions.

Amnesia.—In the field of memory the consciousness of the hysteric undergoes a limitation analogous to the contraction of the visual field. In some instances the loss is slight, the memory of a person or place is lost, or again a complete group of sensory or motor images disappears. A hysteric may suffer from aphasia or agraphia, or may be unable to stand or walk (astasia-abasia). In other instances the loss of memory may be almost complete, and the patient recalls only the facts and ideas closely associated with the mental state of the moment. From the interaction of this amnesia with the susceptibility to suggestion arise the phenomena of double or multiple personality which have been so frequently exploited with relation to hysterics. The defective memory is also the explanation of many of the charges of deceit or wilful lying made against these patients.

Aboulia.—(Loss of will-power.) From this spring vacillation, lack of initiative or enegy, and frequently inability to perform the slightest task. While these three elements, suggestibility, amnesia and aboulia, offer the best explanation psychology can present for the mental phenomena of hysteria, one must admit that they do not satisfy us. Particularly do they fail to account satisfactorily for the behavior of many hysterical persons, whose apparently wilful simulation, deception and falsehood have led many physicians to emphasize their complete moral perversion. Certainly in dealing with them one must beware of being deceived or misled.

Accidents.—The accidents of hysteria constitute the temporary or transitory phenomena of the disease. The accidents are the most striking features of the condition, the symptoms which demand attention and most often bring the patient under the physician's observation. The most impressive of these accidents are the hysterical convulsive attacks. Of these major and minor forms may be described.

Major Form.—The grand attack, as described by Charcot, consists of several phases. (1) A prodromal stage marked by mental depression or exhilaration sometimes accompanied by hallucinations. An aura follows, usually a painful sensation rising from the abdomen to the throat, where it gives rise to a sensation of suffocation, the classic globus hystericus. (2) Convulsive stage. Closely resembling a true epileptic seizure with tonic and clonic convulsive movements. (3) Period of clownism, made up of a phase of contortions, followed by grand movements, such as flexion of the trunk or of the head. (4) Passional attitudes, expressive of fear, love, rage, etc. (5) Period of delirium with hallucinations. Visions are seen, voices heard, conversations held. After

a varying time the hallucinations fade and consciousness returns. Attacks of such elaborate evolution are rarely seen in the United States. We are, however, familiar with the minor or modified attacks.

Minor Attack.—This is regularly preceded by immoderate laughing or crying, and the globus hystericus may be felt. With these prodromata the patient passes suddenly into a convulsion of both tonic and clonic phases resembling the epileptic convulsion. In these seizures it is notable that the patient does not fall suddenly or in such a way as to injure himself or herself; the tongue is not bitten, the urine is not passed involuntarily. The patient's movements are exaggerated and disorderly, so that upon the inexperienced the fit is always more impressive than a true epileptic attack. After a few minutes the convulsion usually subsides, the patient again becomes emotional and returns to consciousness or sinks into a torpid semi-conscious condition which lasts for hours, and from which he or she cannot be easily roused.

Motor Accidents.—P a r a l y s e s .—These occur most often in the form of a paraplegia, but hemiplegia and monoplegia may be met with. The paralysis may be accompanied by anesthesia, or hyperesthesia, and may be as variable in degree as these symptoms. Vasomotor or trophic disturbance is rare, and tendon and electrical reactions are normal. Such paralyses may last days or years.

C o n t r a c t u r e s .—These are closely related to the paralyses. They may be paraplegic, hemiplegic or monoplegic in type. The affected muscles are tense and hard, the part affected rigid, the contractures persist during sleep, but yield to complete anesthesia. Trophic disturbances are rare. Tendon and electrical reactions persist. They may be associated with disturbances of sensation. Rhythmic spasms are not uncommon in hysteria. There may be rhythmic movements of head, face, trunk or extremities, the movements repeated over and over for minutes or days. Tremors are also frequent, either alone or associated with other motor accidents. Any type of organic tremor may be reproduced by the hysterical affection.

Cutaneous Accidents.—Eruptions of various kinds, edema, even gangrene may occur. The phenomena of dermographism can usually be elicited. Hemorrhages from the skin and even bloody sweats are described.

Cardiovascular.—The heart action is usually normal, but may be greatly varied. Rapid heart action and palpitation may give rise to great distress. Slowing of the pulse is much less frequently seen. Attacks of pain referred to the heart and associated with palpitation may closely simulate angina pectoris.

Temperature.—Hysterical fever of various types and heights has been reported. As a rule the pulse and respiration do not correspond. Exceedingly high temperatures have been reported, 112° to 120° F., but suspicion, of course, always attaches to such cases, for no rational explanation of the observations has been given.

Sensory Accidents.—Headache is often severe. It may be referred to the eye, to the vertex or the occiput. It usually comes in attacks in

the afternoon or evening, and may be accompanied by nausea and vomiting. Tenderness over the spine is frequent, and may be so severe as to suggest disease of the vertebræ. Visceral neuralgias are common, and, as already observed, may suggest organic disease of abdominal organs. Hysterical angina pectoris is frequently met with.

Visceral Accidents.—R e s p i r a t o r y .—Aphonia is a common complaint. Mutism is not so often seen. Hiccough, sighing, crying or talking, which may persist for long periods, are well known. Dyspnea, which may be very extreme, the respiration mounting to eighty or one hundred per minute, occurs. Any one of these respiratory disorders appears alone or they may be associated with one another. They arise from sensory or motor disturbances in the respiratory muscles and organs.

D i g e s t i v e .—Anorexia nervosa is the most prominent of these affections. Food may be refused until an extreme emaciation or even death results. Spasms of the esophagus simulating stricture occur. Chronic vomiting may be of hysterical origin. Tympanites may develop and associated with spasm of abdominal muscles may simulate pregnancy (pseudocyesis) or other forms of abdominal tumor. Hysterical peritonitis, that is, hysteria presenting the clinical picture of peritonitis, is well known.

U r i n a r y .—Reduction of the amount of urine is common, complete anuria for long periods possible also, although deception on the latter point must be guarded against. Frequent urination and polyuria are also common, especially at the conclusion of the hysterical attack.

Course and Prognosis.—Hysteria is essentially a chronic affection, lasting for months or years. The patient presents long periods of improvement only under some unfavorable influence, such as undue emotion, or the exhaustion of acute disease to return to the old condition. On the other hand, the various accidents of hysteria, the affections which most often come under the physician's care, are usually readily relieved by appropriate medical treatment. There seems no doubt that hysteria while as a rule not dangerous to life may be fatal through some of its accidents, such as the anorexia and vomiting.

Diagnosis.—The important point in this regard is to be on the lookout for hysteria, as there are few diseases, either acute or chronic, which may not be suggested by the phenomena of this disease. Hysterical affections have ear-marks, which, as a rule, readily identify them. The clinical picture is overdrawn or paradoxical. Thus the hysterical convulsion is a very exaggerated picture of the epileptic attack, and yet the condition of the patient afterward is very much better than in convulsions of organic origin. Anesthesia of hysteria never corresponds exactly to the territory of any nerve or any segment of a cord. The disability produced by hysterical paralysis is always much more marked than in an organic paralysis. The vomiting of hysteria is more severe than that produced by any gastric affection. The hyperesthesia of the abdomen may be more intense than that of an acute peritonitis, so that the suggestion of a touch leads to shrinking and complaint of pain.

This exaggeration of the clinical picture or contradiction between the symptoms and the discernible lesion should always put one on guard. Then an investigation of the history and careful examination of the patient for the stigmata of the disease will lead to a correct diagnosis. On the other hand, it must be remembered that hysterical patients may suffer from organic lesions which pass unnoticed in the presence of hysteria. Thus hysterical patients may easily develop tuberculosis or Bright's disease which may be overlooked.

Treatment.—From the nature of the malady, the treatment of hysteria must be psychic. Suggestibility being so important a feature of the mental condition, an entire change of surroundings and companions is necessary. For this reason complete isolation of the patient is desirable. The visits of family or friends filled with sympathy and ready to discuss the patient's various complaints serve to maintain or even accentuate the perverted mental state. Having secured proper surroundings, the physician must seek to learn the exact mental state of the patient, especially to divine the controlling idea or ideas, and then to meet these by appropriate treatment. Thus the passage of a stomach tube by demonstrating the permeability of the esophagus may relieve difficulty in swallowing. Or by distracting the patient's attention one may be able to bend a previously rigid and powerless limb, and so convince the patient of the possibility of doing likewise.

For the hysterical convulsion a sudden dash of cold water on the face and neck, a sharp command, a pressure upon a hysterogenic zone may bring relief. The common hospital practice of spraying the face and neck with a well-charged bottle of vichy or carbonic water is very effective.

To meet the graver problems of the mental state underlying these accidents demands the utmost skill, tact and perseverance of the physician. Progress can only be made as a rule by gradual education of the patient. For this purpose isolation and the rest cure of Weir Mitchell (see p. 579) have found general favor. But the rest cure can do little for these patients unless seconded by clear insight and the influence of a strong mind. Weeks or months may be required for definite progress. Hypnotism may be of service in some cases, but its use is not unattended by danger, and of recent years has found less and less favor.

Attention must be given to the nutrition and the general health. Anemia, constipation, etc., are to be treated appropriately. Sedatives, such as the bromides, valerian, asafetida, may be used in periods of special excitement or unrest, but altogether medicines have very little value except as called for by some definite symptom. They do not materially affect the underlying conditions.

NEURASTHENIA

Definition.—Neurasthenia is a functional nervous disorder dependent upon exhaustion of the central nervous system, and manifesting itself by symptoms of either general or local weakness and irritability.

Etiology.—Neurasthenia may be either hereditary or acquired. (1) Hereditary. Children of parents who either by reason of scant inheritance or their manner of life have exhausted their store of nervous energy are liable to become the victims of neurasthenia. They have so small a stock of nervous energy to begin with that it may be readily exhausted. (2) Acquired. Neurasthenia is acquired by lack of proper relation between one's store of nervous energy and its expenditure. In normal life each day's damage to the nervous system is made good by the normal process of rest and repair. Whenever the drain becomes too great to be made good by these reparative processes nervous exhaustion or nervous bankruptcy must sooner or later result. Thus prolonged excessive mental labor frequently leads to neurasthenia, and the disease is common among overworked business and professional men. On the other hand, neurasthenia may be seen following the exhaustion of some of the acute infectious diseases, such as syphilis, typhoid fever and especially influenza. Finally, overindulgence in alcohol, tobacco, opium or cocaine may be the underlying cause for the development of neurasthenia.

Inasmuch as the explanation of the disease is not infrequently a congenital lack of nerve force, it is not surprising that we at times meet with neurasthenia in children. It becomes much more frequent at the period of adolescence, especially in girls, when the normal demands of development as well as those of education put an unusual tax upon the nervous system. It is most frequently seen in mature life among men and women overborne by the cares of business or the worries of society.

Symptoms.—The symptoms of neurasthenia are those of a slowly but steadily increasing exhaustion of either mind or body, or both. In the beginning the patient finds himself more easily exhausted by either mental or physical effort. The day's work calls for greater exertion than usual. He is more nervous and more easily disturbed by the ordinary cares of life. As the process goes on he slowly but surely develops the full picture of nervous prostration. In its fully developed form the symptoms of neurasthenia may be classified as psychic, sensory, and motor or somatic.

Psychic.—The psychic phenomena of neurasthenia are essentially those of brain fatigue. The patient can no longer carry on his simplest duties without an unusual degree of mental exhaustion. The most ordinary form of mental effort may become impossible. Reading, writing, even ordinary conversation, may become too great a task for the weary brain. From this mental exhaustion of the brain, loss of will power, lack of initiative, inability to concentrate the attention, impairment of memory, and altogether a more or less complete failure of mental power result. Loss of the power of inhibition leads to an abnormal irritability. The ordinary trials of life affect the victim unduly, and he frequently feels himself misjudged and ill-used. Anxiety is always a prominent feature of the mental horizon of the neuras-

thenic. He worries both about his affairs and his physical condition. He is sure that some great disaster threatens him or that he is about to develop some deadly disease. In the extreme cases this anxiety of mind may become an abiding fear, producing the so-called phobias of the disease, such as agoraphobia or the fear of open spaces, or monophobia, fear of being alone, claustrophobia, the fear of being shut in, or as the climax of all, pantophobia, the fear of everything. The mind may be so harassed by fear and anxiety that suicide is not out of the question in these cases.

Sensory Symptoms.—Headache is an almost constant concomitant of neurasthenia. The headache is regularly referred to the occiput, and is described in varying terms as a sense of oppression and constriction or dull or sharp pain. The headache may be referred to any part of the head and may be described in various ways. Usually associated with headache, there is a sense of lightness or fulness in the head, and vertigo of a more or less intense character is not infrequently met with. Pain in the back is regularly associated with the headache. This pain is most frequently felt in the lumbar region, but it may be referred to any part of the spine. With it there may be points of tenderness scattered up and down the back over the spine, or under the angles of the scapulæ. Pain and disturbances of sensation of any and all kinds may be met with in any part of the body.

Pain may be complained of in any part of the body, and sensations of heat, cold, prickling, tingling, or numbness are common. On the other hand, anesthesia does not occur in uncomplicated neurasthenia. The most careful local examination fails to detect any adequate cause for the complaint of pain or disturbance of sensation.

The Motor Symptoms.—The muscular weakness of neurasthenia is often pronounced. The most ordinary exertion, such as walking a few blocks, may produce profound fatigue and complaint of pain. Effort that was formerly easy becomes impossible, the patient may even take to bed on account of physical disability. The underlying muscular fatigue may easily be demonstrated by asking the patient to repeat any voluntary muscular act, such as compressing the dynamometer. The action may at first be well executed, but each returning effort shows a rapid loss of muscular power. This myasthenia may be accompanied by tremor of the hand or face or local twitching. The tendon reflexes in neurasthenia are regularly increased, and a slight ankle-clonus may be demonstrated in some instances. Frequent repetition of these reflexes will, however, demonstrate the underlying muscular weakness by rapid exhaustion.

Somatic Symptoms.—Neurasthenic patients often lose weight and become anemic. In the severest cases the malnutrition may be extreme. On the other hand, abundant adipose tissue and good color are not inconsistent with the diagnosis of neurasthenia. Apart from the general bodily disturbance, neurasthenia results in affections of individual organs, marked by excessive irritability, which is often characteristic.

(a) Thus irritability of the retina or the muscles of the eye may give rise to painful sensations in the eyes and forbid prolonged use. Irritability of the retina may produce marked sensitiveness to light and even photophobia. (b) In like manner the auditory functions are disturbed, and there may be abnormal sensitiveness to noise of any kind. (c) Cardiac palpitation is one of the most common symptoms of neurasthenia, and often the cause of great discomfort. It is closely related to the digestive disturbances which belong to the disease, but may occur independently of them. Frequently there is associated with it a painful throbbing of the arteries, most notably seen in the abdominal aorta, but extending even to the peripheral vessels. (e) More or less disturbance of the digestive functions regularly accompanies neurasthenia, and in some instances this digestive disturbance may constitute the chief feature of the clinical picture. The patient complains of considerable discomfort after eating, and there is a more or less constant belching of air. Similarly the functions of the intestines are impaired, and constipation regularly accompanies the disease. Sexual disturbances frequently develop in the neurasthenia of men. They are distressed by nocturnal emissions or spermatorrhea, and suffer greatly from fear of impotence.

Insomnia is one of the most constant symptoms of neurasthenia. There may be difficulty in getting to sleep or the patient after falling quietly asleep is disturbed by dreams, is restless, and awakes unrefreshed in the morning.

One may find various types of neurasthenia described, such as cerebral, spinal, gastric or sexual, depending on the prominence of symptoms referred to these several parts, but neurasthenia is essentially a disease of the entire nervous organism with varying emphasis on symptoms referred to special organs.

Course.—The onset of neurasthenia is insidious, gradual. Once established the neurasthenic symptoms persist indefinitely unless interrupted by some radical change of life or proper treatment. The disease may be protracted for years with varying periods of improvement and relapse. Patients who have once suffered from neurasthenia are liable to recurrence when subjected again to any unusual nervous strain.

Diagnosis.—The picture of mental and physical weakness, combined with the impairment described in the functions of the various organs, is characteristic of neurasthenia. The essential feature of the disease is that of irritable weakness. There is no actual loss of power, such as is seen in a paralysis or contracture of organic origin. The most important part in the diagnosis of neurasthenia is to take care by thorough physical examination to exclude the possibility of organic disease, of which the neurasthenia may be purely symptomatic.

Prognosis.—Neurasthenia is rarely or never fatal except from suicide. Recovery is the rule, if proper treatment can be instituted and carried out. The younger the patient the more likely he is to recover,

but the more liable he is to relapse. In patients over the age of forty the disease is very likely to become chronic.

Treatment.—The prophylaxis of neurasthenia in childhood involves the adjustment of the life to the physical and mental constitution of the individual. The problems of diet, discipline, time of entering school, school hours, and school studies, rest, recreation and amusement must all be settled in such manner that, whatever the demands upon the nervous system of the child, they shall not only be met, there shall be laid up a store of nervous energy for use in later years. There is no question that many children suffer from defective management in this respect. The long hours at school with the heavy tasks at home often work serious harm. Far too many children during their school careers present manifest evidences of neurasthenia, or at its close are so sapped of nervous energy that they have no store left to meet the demands of later life. The prevention of such results should be part of the duty of every family physician. The prophylaxis of neurasthenia in middle life calls for exactly the same adjustment of the nervous energy of the patient to the demands made upon it. At this time we must take into consideration the nature of the occupation, the amount of time devoted to it, the manner of life, the question of rest or recreation and amusement, every phase of life calling for the expenditure of nervous or physical energy, and the means of restoring them. Inasmuch as at this period the disease is essentially a fatigue neurosis, the vital point in its treatment is to secure rest for the nervous system. In the milder cases a temporary reduction of work, combined with exercise in the open air, and freedom from care, may be all that is required. In other cases a good vacation in the woods or a trip abroad may enable the patient to regain his lost nerve force. In the severer type of the disease, however, adequate treatment requires months of time, and taxes the skill of the most resourceful physician. The patient often requires physical as well as mental rest, and isolation is necessary. For such patients the rest cure of Weir Mitchell has proved the most satisfactory method of treatment. In addition to isolation, rest in bed, overfeeding, massage or electricity are used, all for the purpose of restoring nerve and muscle tone, or for suggestive impression. The general features of this method are presented in the following details of the daily program:

Seven A.M., cocoa, followed by a cool sponge bath with a rough rub, and the toilet for the day. 8 A.M., breakfast with milk. Rest an hour after. 10 A.M., 8 ounces of peptonized milk or its equivalent. 12 M., 8 ounces of milk or soup, after which the patient can be read to by the nurse. 1.30 P.M., dinner, followed by rest for an hour. 3.30 P.M., 8 ounces of milk and a half hour later the application of electricity. 6.30 P.M., supper with milk, followed by rest for an hour, after which the patient may be read to for half an hour or longer, if it does not fatigue him or her. 8.30 P.M., administration of malt extract with an aperient, if necessary, or the utilization of some measure of treatment for insomnia, such as the drip sheet, or the administration of an hypnotic. Valuable

as this method is in itself, it will succeed only when backed by the constant service of an intelligent nurse and the influence of a physician who has faith in it, and who enjoys the confidence of his patient. Under the treatment patients regularly gain rapidly in weight and in nervous tone. The treatment must be carried out for weeks or months, depending upon the severity of the case, and the progress made toward recovery.

The relief of local sources of irritation is not infrequently an important part of treatment. Thus the wearing of proper glasses for the relief of eye-strain may be an essential element of the cure. Similarly the proper treatment of uterine or ovarian disease may in some cases be very important, but radical operations upon neurasthenics are to be avoided as long as possible. Finally alcoholism or drug habits must be treated by appropriate measures.

In other and milder cases, especially in men, systematic physical exercise in the open air is of great value. In the early stages an hour's work in a gymnasium daily, or every other day, allays the disturbance and permits continuance of work. In severe conditions weeks or months must be given up to systematic physical culture under ideal conditions.

Physical culture to be most effective must be combined with complete relief from mental labor or anxiety, and with such diversions as employ the time and lead to entire peace of mind.

Hydrotherapy is of very great value. Spinal douches, cold packs, or cold shower baths, etc., must be employed according to the patient's condition. Massage is invaluable for the relief of the general physical exhaustion or for local irritability.

Electricity is often employed for its sedative effect upon the spinal centers especially, and the various forms of electric current, high frequency, sinusoidal and the Roentgen rays, are employed for the purpose. Various sanatoria and health resorts employing some or all of these measures offer the most effective treatment for these patients.

TRAUMATIC NEUROSES
(Traumatic Neurasthenia or Hysteria)

Definition.—A condition of neurasthenia or hysteria developed as a consequence of the physical or psychic effect of accident or injury.

Etiology.—Any mental shock may be followed by the development of neurasthenia or hysteria. Physical injury increases the probability of such a result. Neurasthenia or hysteria therefore frequently follows accidental injuries sustained in railroad accidents, fires, explosions and the like. They may, however, follow when no injury has been received, merely from the mental shock. The possibility of recovering damages often intensifies the effects of the experience.

Morbid Anatomy.—Many theories have been advanced as to the nature of the changes underlying these conditions, but none of them has been satisfactorily demonstrated.

Symptoms.—The clinical picture is that of neurasthenia or hysteria, or a combination of the two. It may include any of the usual symptoms of these disorders, even in their severest forms. Pains of various kinds and locations, tingling, numbness, formication and the like, and loss of power, complete or partial, hemiplegic, paraplegic or monoplegic in type, are common complaints. Mental power, speech and the special senses may suffer in an endless variety of ways.

Course and Prognosis.—The conditions usually improve slowly, and health is ultimately regained. Recovery is often prompt after decision of damage suits, because of the mental relief, and quite independently of the question of malingering. In some cases the condition becomes chronic and persists indefinitely.

Diagnosis.—Careful investigation of the history of the patient and careful physical examination are essential. Previous organic disease may be excluded by these means, for it must be remembered that patients having organic disease may present hysterical symptoms. Malingering must be borne in mind and the patient subjected to various tests to bring out the sincerity of his complaints. Thus spots reported exceedingly painful and tender may be freely pressed, if the patient's attention is diverted by conversation, or the reflexes of a supposedly paralyzed limb found to be normal. Hoover's test for hysterical paralysis of the leg is of interest. In organic hemiplegia, if the patient lying supine attempts to raise the paralyzed leg the heel of the normal side is pressed downward—*i.e.*, into the bed. In hysteria or malingering no such action would occur.

Treatment.—The treatment must be that of neurasthenia or hysteria.

PERIODIC PARALYSIS
(Family Periodic Paralysis)

Definition.—A flaccid paralysis of the muscles of the trunk or extremities, temporary and recurrent at fairly regular intervals.

Etiology.—The disease appears in certain families, in one recorded instance affecting four successive generations. The transmission is by either male or female branch. Cases have been recorded as early as the fifth year, but the onset may be delayed till after thirty. The pathology of the disease is not established. A certain resemblance to myasthenia gravis is noted.

Symptoms.—Over night, the patient, previously well, develops a flaccid paralysis of all extremities, and possibly of the trunk. The reflexes are lost. In some instances even the muscles of the face and tongue have been affected.

The paralysis lasts hours or days, and then gradually disappears. Milder and abortive attacks may be observed. The attacks recur from time to time without apparent cause. The severity of the attacks diminishes later in life and death is rare. Treatment must be directed to the maintenance of normal physical and mental tone. The systematic administration of the bromides is advised.

ASTASIA-ABASIA

These terms denote a certain symptom-complex met with in various conditions, especially hysteria, epilepsy, chorea and the like. Although muscular power, sensation and coördination are normal, so that lying in bed power and motion are normal, the patient is unable to stand (astasia) or to walk (abasia). Most cases show no spasm, rigidity or ataxia; in others there are spasms and irregular jerky movements. The treatment must be directed to the underlying condition.

RAYNAUD'S DISEASE

Definition.—A vasomotor affection, showing no organic lesions, in which localized anemia or congestion leads to disturbance of function or rarely to necrosis of the part involved.

Etiology.—The disease is rare. In many cases no explanation is to be found. In others a hereditary or family neurotic constitution is accused. Sudden fright or shock may precede the attacks. Women are attacked more frequently than men, and cases in children have been recorded. Exposure to intense cold is often given as the cause of attacks.

Morbid Anatomy.—Theory has attributed the condition to spasm or paresis of the vaso-constrictors in the affected part. Peripheral neuritis has been found in some cases; endarteritis obliterans in others, but these cases with definite organic lesions should, for the present, be excluded from this category.

Symptoms.—Three stages of the disease are described, in any one of which the patient may first be observed.

1. Stage of local syncope. Following exposure to cold, mental shock or excitement or gastric disturbance, one or more·fingers or toes or the whole hand or foot becomes white, cold, and numb. This condition persists for a few hours, and then the circulation returns to normal or passes into the second stage.

2. Stage of asphyxia. The affected part or parts become livid, mottled, deep red or purple. Intense pain or anesthesia may be complained of. The parts may appear swollen and puffy. Like the first stage this condition persists for some time, and then gradually gives place to the normal. The affection is frequently symmetrical on hands or feet. In most cases the attacks are repeated from time to time, either with or without definite cause.

Constitutional symptoms are ordinarily lacking, but hemoglobinuria has been observed in some patients, as though hemolysis had occurred, and in others temporary visual disturbances, mental dulness, hemiplegia or aphasia during the attacks have suggested that vascular disturbances similar to those in the extremities were taking place in the eye or the brain.

3. Stage of gangrene. Occasionally a dry gangrene follows the cyanotic stage. The affected finger or toe becomes dry, shrivelled, dead; a line of demarcation forms in time and the dead portion sloughs away.

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Slow cicatrization follows. A hand or leg may be lost in successive attacks. The ears or nose may also be involved in severe cases.

Diagnosis.—The phenomena of Raynaud's disease are easily recognized. The propriety of the diagnosis depends upon the careful exclusion of definite organic disease. Error is easy in patients seen only once. The recurrence of the phenomena is highly suggestive of Raynaud's disease.

Diabetes, embolism or thrombosis of the vessels of the affected part, disease of the spinal cord, such as tabes or syringomyelia, multiple neuritis (beri-beri) and ergot-poisoning must be excluded by care in the examination of the patient. Of late the studies of Buerger and others have laid emphasis, especially in elderly patients (Hebrews), upon the presence of a definite obliterating thrombosis in the arteries of the affected extremity.

Treatment.—The general health of the patient must be maintained on as high a level as possible. If cold causes attacks, residence in a warm climate during the winter is advised. Hydrotherapy, local massage, and electricity, either galvanism or the high frequency current, have been of service.

During the attacks pain may be relieved by bathing in hot water, or the hot-air bath. Bier's method may be tried, if other measures fail. In severe cases morphine may be required. If gangrene develops, the part must be kept as nearly aseptic as possible. Amputation may in the end be necessary.

ERYTHROMELALGIA
(Red Neuralgia)

Definition.—A rare chronic vasomotor affection in which congestion, pain and local heat develop in some part or parts of the body, especially in the extremities, when they are dependent.

Etiology.—Men are more often affected than women. Exposure, overexertion and sudden strain have been considered as possible causes. The condition is regarded as a vasomotor neurosis closely related to Raynaud's disease.

Morbid Anatomy.—Local lesions of the arteries, or the nerves, or the posterior spinal nerve-roots have been found, but the pathology is not yet established. A localized neuritis or endarteritis would explain the conditions.

Symptoms.—Redness, pain and swelling develop in the affected part, most often a foot or hand, especially following use. The affection may be symmetrical. Standing or walking intensifies the disturbance. The condition is more or less chronic, but may disappear.

Diagnosis.—As in Raynaud's disease careful examination must be made for organic disease. From Raynaud's disease the affection is distinguished by its prevalence in women, the absence of any stage of anemia, the increase of symptoms when the part hangs down, or on exposure to heat (summer), the higher temperature of the affected parts,

and the absence of gangrene. In some cases the distinction is extremely difficult.

TREATMENT must be along the lines indicated under Raynaud's disease. Cold rather than heat gives relief to pain. Rest is important. Excision of the nerves of the affected part has been helpful.

FACIAL HEMIATROPHY

Definition.—A slowly progressive atrophy of the bones and soft tissues of one side of the face.

Etiology.—About 200 cases of this rare affection are recorded. The patients are usually women. The cause is not known. At autopsy an interstitial neuritis of the fifth nerve has been demonstrated. The cervical sympathetic may also be involved.

Symptoms.—Whitish patches (leukoma) appear on the skin of the face. Skin and subcutaneous tissue atrophy till the skin appears to rest upon the bone. The hair and eyebrows may fall out or become white. The eyeball may sink, owing to disappearance of the orbital fat. The tongue may show hemiatrophy. Even the bones atrophy and the teeth may fall out. The result is a very great disparity in the two sides of the face. Various sensory disturbances may accompany the atrophy.

Treatment is unavailing.

ANGIONEUROTIC EDEMA
(Quincke's Disease)

Definition.—A neurosis characterized by the development of areas of acute edema of the skin or mucous membranes.

Etiology.—Young adults are most often affected. Like other neuroses the tendency appears to be hereditary in some families in which other neuroses are known. Theoretically the condition results from a localized vasomotor disturbance. It closely resembles urticaria and may be related to the skin reactions produced by the infection of antitoxic and other sera, the so-called anaphylaxis.

Symptoms.—Sharply localized areas of soft, puffy swelling develop on the face, especially the eyebrows, ears or lips, on the hands or feet, or on the tongue, throat or genitalia. The swellings are diffuse, smooth, not tender or painful, pale or slightly flushed. There are no constitutional symptoms. The swellings last a few days, then disappear, to recur from time to time, at irregular intervals.

In a few cases constitutional symptoms, headache, fever, and somnolence, accompany the edema.

In patients whose gastro-intestinal mucous membrane is involved, severe attacks of abdominal pain, vomiting and prostration may occur. Death has resulted from such attacks or from acute edema of the larynx of angioneurotic origin. In such visceral attacks the affection is closely akin to Henoch's purpura, the lesions being edema instead of hemorrhages.

Treatment.—The general health must be improved by attention to diet, exercise, rest and hygiene. Articles of diet found to produce attacks must be excluded. The administration of calcium lactate, 20 grains, thrice daily, may be helpful.

SCLERODERMA

Definition.—A diffuse or patchy induration of the skin sometimes followed by atrophy, apparently a trophoneurosis.

Etiology.—The patients are commonly between 20 and 40 years of age; two-thirds of the cases recorded have been in women. The affection may follow an acute infectious disease, but there is no satisfactory etiology.

Morbid Anatomy.—Endarteritis of the vessels of the affected area with hypertrophy of the several parts of the skin, but especially the connective tissue, has been found in most cases examined.

Symptoms.—The face and extremities are the most common seats of the affection. The affection may remain localized (morphea) or may extend to the skin of the whole body.

A white marble-like induration of the skin is usually the earliest symptom. On the affected area the skin cannot be picked up in the usual fold. It indents but slightly, if at all, on pressure. More or less stiffness of the affected part results. Atrophy of the skin gradually follows, and it then becomes dry, parchment like, and adherent to the underlying tissues. Areas of deeper pigmentation (brownish) and patches of leukoderma are found in the atrophic areas. An erythema or cyanosis of the affected skin is sometimes observed, particularly in the hands or feet, giving the affection a close resemblance to Raynaud's disease. Trophic disturbances, such as paronychia, ulceration, and loss of hair, may occur in the skin itself, and in the underlying parts an atrophy of both muscle and bone has been observed. Constitutional symptoms, such as fever and diarrhea, have been recorded, but are rare.

Course and Prognosis.—The disease may run a rapid or a chronic course. Some cases recover, many die of exhaustion.

Diagnosis.—The affection is usually easily recognized. Raynaud's disease is suggested by some cases, leprosy by others. Myxedema must also be considered.

Treatment.—The continued administration of the thyroid extract has seemed to help some cases. Many other drugs have been tried but without result. Hydrotherapy, especially as carried out at various mineral springs, the local application of the Roentgen rays, massage and electricity are recommended.

ACROMEGALY

Definition.—A rare chronic disease marked by progressive increase in the size of the bones of the head and extremities, and related to morbid function of the pituitary gland.

Etiology.—The disease develops in the third decade. People of large size are especially subject to it, but dwarfs are not immune. No satisfactory cause is known.

Morbid Anatomy.—All the bones of the body, but especially those of the extremities, are enlarged. The increase is due to subperiosteal growth. All prominences, ridges, or grooves are accentuated. The outer table may be increased in density. Exostoses are common. The pituitary gland is often enlarged, especially the anterior lobe. The enlargement may be of any pathological type, simple hypertrophy, a cyst, or a malignant growth. Enlargement of some of the thoracic organs has also been observed.

Symptoms.—The patients complain of headache, irritability, loss of memory, pains in various parts of the body, constipation, and polyuria. On examination the characteristic overgrowth of the head, the nose, the jaws and the extremities is found. The larynx is often enlarged and the voice is deeper than normal. The spine shows various deformities, kyphosis, scoliosis, or curvatures. The hands and feet may be enormous. All parts of the body, orbit, ears, heart, liver, and spleen may show enlargement.

The muscles are not affected and reflexes are normal. An unlimited variety of disturbances of the functions of the body has been recorded in various cases.

Acromegaly often presents some of the symptoms of exophthalmic goitre, syringomyelia, myxedema or epilepsy.

THE COURSE of the disease is variable. Remissions occur and the course is very chronic, lasting even fifty years. Malignant cases with very rapid enlargement are rare. A cachexia finally develops and the patients die of exhaustion or from the dilatation and failure of the heart.

Diagnosis.—In typical or advanced cases the diagnosis is easy. In early stages the enlargement of the extremities may be overlooked and the disease mistaken by reason of the variety of associated symptoms. Skiagraphs of the bones of the head and extremity are important. Enlargement of all these parts belongs only to acromegaly. Pulmonary osteo-arthropathy gives a history of pulmonary disorder and no involvement of the face, and no nervous symptoms. Gigantism presents no disproportionate enlargement of the bones. Arthritis deformans and osteitis deformans may be suggested, but are readily excluded by careful examination.

Treatment.—Pituitary therapy has thus far been without value. If a definite tumor of the pituitary can be made out, surgical removal may be attempted. Otherwise treatment must be wholly symptomatic.

THE GENERAL CARE OF THE SICK

The adequate treatment of the sick involves much more than the prescription of medicines. In many of the conditions with which we have to deal the welfare of the patient depends more upon the general régime, rest, careful feeding, regular evacuation of the bowels, and sleep, than upon the medicines administered. While these general subjects have been mentioned in the discussion of the treatment of diseases already given, certain details require separate presentation.

Choice of the Sick-room.—In most instances other considerations than choice determine the question of the character of the sick-room. Where choice is possible the room should be large, at least 1,000 cubic feet in capacity, and should have abundance of sunshine and fresh air. In certain conditions, such as meningitis or measles, it may be necessary to darken the room, but under ordinary conditions both light and air are to be freely admitted. The light should, however, never fall directly into the eyes of the patient.

Ventilation.—For many years all attempts to ventilate sick-rooms or hospitals were based upon calculations of the amount of carbon dioxide given off by one person and the atmospheric content of this gas consistent with comfort and health. Thus it was calculated that an individual exhales 14.4 cubic feet of carbon dioxide in 24 hours, and that the proportion of this gas in the air should not exceed six parts in 10,000. To meet these conditions it was established that each individual requires 3,000 cubic feet of fresh air per hour. On this basis many different systems of ventilation for homes and hospitals have been devised, but none of them has proven equal to the free ventilation secured by opening windows wide or placing the sick on balconies or roofs, or entirely in the open air. The success attending the open-air treatment of the tuberculous has led to the adoption of the method, modified to meet different conditions, in the treatment of most of the disorders included within the sphere of internal medicine. The most satisfactory method in hospitals consists in having balconies in direct connection with the wards, so that the beds mounted on casters may be wheeled into the open air. For purposes of treatment requiring the exposure of the body of the patient, the bed is taken into the ward. Experience with this method has proven that so long as the body and especially the extremities are kept warm, even the feeblest patients are invigorated by breathing cold fresh air. Draughts have been shown to be harmless, if the head be protected by cap or hood. Thus protected, even patients suffering from pneumonia may lie in the draught of an open window with positive advantage. Chilling of the surface of the body should at all times be avoided by adequate bed-covers or clothing, especially in

patients suffering from circulatory disorders with heightened blood pressure, since the contraction of the peripheral vessels by cold may increase the pressure. In the feeble the condition of the feet should be constantly watched, and, if they tend to become cold, artificial heat should be applied, usually by the use of the hot-water bottle. So long as the external atmosphere is cold, the hands and arms should be kept under cover. If this is impossible, on account of restlessness or delirium, they should be protected by capes and mitts.

In private houses it is ordinarily necessary to close and warm the sick-room for baths or any other treatment requiring the exposure of the body, since the difficulty of moving the patient from one room to another is too great.

In applying these teachings it must be borne in mind that they are radically opposed to many deep-rooted convictions of the layman. The fear of fresh air, and especially of draughts, is still common, and only by judicious management can the physician who attempts to carry out the open-air treatment of his patients escape criticism or censure for developments not justly chargeable to the method.

Cleaning the Sick-room.—Whenever practicable the patient should be removed to another room. In any event the room should be cleaned and dusted in such a manner that dirt and dust will be gathered up, not scattered abroad. For this purpose vacuum cleaning is ideal. Sweeping should be done with a broom covered by a moistened cloth, and the dusting-cloths should likewise be moist.

Isolation in Contagious Diseases.—In the treatment of contagious diseases, measles, scarlet fever, diphtheria, small-pox, German measles, chicken-pox, etc., special care is necessary to prevent the spread of the infection. The regulations of the New York Board of Health for the isolation of cases of measles, scarlet fever or diphtheria are here given: 1. Complete isloation of every case of diphtheria as ordered by the medical inspector of the Department of Health must be maintained until the disease is at an end and fumigation has been performed. 2. Children in the family must not be allowed to attend school until they have received a certificate from the Department of Health. 3. The room used for the case should be as nearly bare of furniture as possible. Carpets and hangings should be removed before the patient is placed in the room. Toys or books used by the sick person should be thoroughly disinfected or destroyed after recovery or death. The sick-room should be well aired several times daily, the floor mopped and woodwork frequently wiped with damp cloths. Under no circumstances must the floor be swept when it is dry. It should be sprinkled with sawdust, bits of newspaper or tea leaves, all thoroughly moistened, and then carefully swept so that no dust may arise. 4. When practicable, one attendant should take entire care of the patient, and no one else beside the physician should be allowed in the room. The attendant should have no communication with the rest of the family. Visitors must not be ad-

mitted to the apartment as long as the placard remains on the door. 5.
Plates, cups, glasses, knives, forks, spoons, etc., used by the patient
should be kept for his especial use, and under no circumstances removed
from the room or mixed with similar utensils used by others. They
should be washed in the room in hot soap-suds, and then rinsed in boil-
ing water. After use the soap-suds should be thrown into the water
closet. 6. All cloths, bed linen and personal clothing which have come
in contact in any way with the patient should be immediately immersed
in a $2\frac{1}{2}\%$ carbolic solution before removal from the room. They
should be soaked for one hour and may then be removed from the room
and boiled in water and soap-suds for five minutes. 7. Surfaces of any
kind soiled with discharges should be immediately washed with the car-
bolic solution. 8. The discharges from the nose and mouth of the
patient should be received on handkerchiefs or cloths, which should be
at once burned or immersed in a $2\frac{1}{2}\%$ carbolic solution. 9. After mak-
ing applications to the throat or nose of the patient, and before eating,
the hands of the attendant should be disinfected by scrubbing in hot
soap-suds. 10. When the skin of the patient is peeling, the body should
be washed daily with warm soap-suds and afterwards anointed with oil
or vaseline. This should be repeated until all roughness of the skin has
disappeared. 11. After the inspector of the Department of Health has
ordered fumigation, the entire body of the patient should be bathed and
the hair washed with hot soap-suds. The patient should then be dressed
in clean clothes (which have not been in the sick-room during the illness)
and removed from the room. The attendant should also take a bath
and put on clean clothes before mingling with the family or other peo-
ple. The clothes worn in the sick-room should be left there to be fumi-
gated with the room and its contents. Under no circumstances should
the sick-room be again entered or occupied or anything removed from
it until fumigation has been performed.

In the milder contagious diseases, such as chicken-pox, German
measles, whooping-cough, the isolation is less rigidly practiced, but
always with some risk to others.

The source of contagion in cholera, dysentery and typhoid fever
being limited to the feces and urine, the spread of the contagion can be
prevented without isolation, but cholera patients, on account of the fear
of the disease, are usually isolated, and isolation of typhoid fever
patients has been repeatedly urged. Visitors or others coming in con-
tact with the bed clothing of such patients are certainly exposed to dan-
ger of infection.

It would hardly seem necessary to forbid the kissing or fondling of
patients sick with contagious diseases, but experience teaches the need
of constant repetition of such prohibition.

Disinfection.—The room or rooms occupied by a person suffering
from contagious disease should be disinfected. This is usually required
by Boards of Health after small-pox, measles, scarlet fever, diphtheria,

cholera, the plague, typhus, typhoid fever and tuberculosis. It is desirable after the milder affections, such as chicken-pox, German measles and whooping-cough.

In every case of disinfection in New York City the following regulations must be complied with:

All cracks or crevices in rooms to be disinfected must be sealed or calked, to prevent the escape of the disinfectant.

The following disinfectants may be used in the quantities named:

Sulphur, 4 lbs. for every 1,000 cubic feet, 8 hours' exposure.

Formalin, 6 oz. for every 1,000 cubic feet, 4 hours' exposure.

Paraform, 1 gr. to every cubic foot, 6 hoars' exposure.

After fumigation for diphtheria or scarlet fever, bedding, carpets, rugs, etc., will be removed for sterilization. For this purpose formaldehyde and steam under pressure are employed.

All glass or earthenware dishes or vessels used about the patient should be soaked for several hours in 5% carbolic or 1/1000 bichloride solution. Metallic objects should be treated with carbolic. The woodwork should be wiped with the carbolic solution and the walls should be freshly papered or painted.

General Care of the Patient.—THE SKIN.—A daily cleansing bath is desirable. If the patient's condition permit, he may be allowed to take an ordinary tub-bath. If he is confined to the bed, a sponge-bath may be given by a nurse or attendant. After such a bath it is the common practice to rub the body, but especially the back, with alcohol, and follow this with a simple dusting powder of talcum or boracic acid. This use of alcohol and powder must be repeated several times during the day in the seriously sick. It is not only grateful to the patient but serves to prevent the furuncles, abscesses or bed sores which so often develop in the bed-ridden, especially in those suffering from lesions of the nervous system attended by paralysis or loss of sensation.

The development of bed sores, which are largely due to the influence of pressure upon tissues whose vitality is lowered by exhausting disease, may be further guarded against by frequent changes in the position of the patient, the judicious use of pillows, air-rings and like devices for taking the pressure off threatened areas. If bed sores develop, they must be relieved of pressure, cleansed antiseptically and dressed with some stimulating application, such as balsam peru and castor oil, equal parts, or a dry powder of boric acid or other antiseptic. Watchfulness is necessary to detect collections of pus about or leading from a bed sore, and by early incision limit the extension of such processes. In any case of protracted illness when bed sores threaten, an air- or water-bed is advisable.

THE EYES.—The bed should be so placed that the light does not fall directly in the patient's eyes. If necessary, an eye-shield may be employed, or the face shaded by a screen. Only under rare conditions, such as acute meningitis or tetanus, or in diseases accompanied by acute

inflammations of the eyes, should the room be continuously darkened. On the other hand, darkening the room for an hour after the mid-day meal is most helpful in enabling a patient to get a nap at that time.

THE MOUTH.—In all fever patients and in those too feeble or indifferent to care for themselves, the mouth must be given special attention. Such patients regularly breathe through the mouth, the mucous membrane becomes dry, brown, fissured, and perhaps eroded, the tongue and teeth are covered by an accumulation of food, desquamated epithelium, bacteria, and in severe cases, blood. Feeding is interfered with by pain and the mouth becomes a source of infection to the adjacent glands and tissues, and possibly of systemic infection.

In these cases, therefore, the mouth must be cleansed after each feeding, either by rinsing with an antiseptic solution, such as the liquor antisepticus or liquor antisepticus alkalinus of the National Formulary. When the patient cannot do this for himself, the mouth should be cleansed by the nurse. For this purpose cotton may be wrapped on bits of wood or upon the nurse's finger, and by these means the solution may be carried to all parts of the mouth.

If the parts become dry, they may after washing be anointed with albolene or white vaseline.

THE HANDS.—Attention to the condition of the hands is very necessary in the severely sick, especially in typhoid fever and other diarrheal conditions. The possibility of reinfections from the hands must be borne in mind, and care taken to prevent them. In delirious patients it may be necessary to tie the hands to the sides of the bed to prevent constant picking at the lips or nose.

THE FEET.—Coldness of the extremities adds greatly to discomfort and vital depression. In the seriously sick or very feeble the condition of the feet should be constantly watched and their warmth maintained by sufficient covers or the use of the hot-water bottle.

ENEMATA.—1. *Cleansing.*—For this purpose water at 100° F. containing sufficient castile or other mildly irritating soap (not laundry soap) to make it turbid is employed. For an adult from one to two quarts are ordinarily given. The patient should lie upon his back, with the hips elevated on a douche pan, but if this position is distressing, the injection can be given with the patient sitting up. The fluid to be injected is suspended in a douche-bag not more than three feet above the level of the body. The ordinary hard-rubber douche nozzle may be employed, but the soft rectal tube is preferable. In introducing the rectal tube care must always be taken to prevent its doubling upon itself in the rectum. After the tip has been inserted its advance is favored by allowing a little of the fluid to enter in advance of the tube. (2) Milk and molasses in equal parts have been found to make a very satisfactory cleansing enema. About eight ounces of the mixture are usually required. The relatively small quantity is in some patients an advantage. (3) For the relief of abdominal distention and tympan-

ites, such as occurs in many acute diseases, especially in pneumonia and typhoid fever, it has been found advantageous to add various drugs to the cleansing enema. An effective combination contains one dram of turpentine, two of ox-gall, three of milk of asafetida to eight ounces of soap-suds.

Any of these cleansing enemata may be reinforced by the addition of half an ounce or an ounce of glycerine, which not only aids in softening the fecal material, but by its hygroscopic effect intensifies the action of the injection.

A distinction is commonly made between a low and a high enema. An injection syringe with a simple hard-rubber nozzle is often employed for the introduction of the fluid, which under these conditions hardly passes beyond the rectum. It has been found more advantageous to have the fluid, as a rule, pass as far into the colon as possible. To accomplish this the patient must be recumbent with the hips elevated, while a soft-rubber rectal tube or large-sized catheter attached to douche-pipe is passed as far as possible into the rectum. There is doubt whether the tube, no matter how long, passes beyond the rectum, but the fact remains that by these means the fluid is carried higher than under the older procedure, and the colon is more thoroughly emptied. Such an enema is commonly termed a "high enema."

2. *Nutrient.*—Various forms of nutritive enemata are employed. The basis of all is milk, from 6 to 8 ounces, to which are usually added one or two eggs and a teaspoonful of common salt. This mixture should be thoroughly peptonized. Some add from one-half to one ounce of lactose or glucose to the mixture. If stimulation is desired, whiskey or other medicine can be given at the same time. Since opium has no local effect, the custom of adding this drug to an enema in the hope of allaying irritability of the rectum is not well founded. A hypodermic of morphine would be much more effective.

One hour before giving a nutritive enema, the rectum and colon should be thoroughly emptied by a high enema, and after it the patient should be kept perfectly quiet for at least an hour.

For the introduction of a nutritive enema a simple funnel attached by not more than a foot of rubber tubing to a rectal tube or large catheter should be used, and no more force employed in its introduction than is necessary. The funnel should be raised just sufficiently to allow the fluid to pass into the bowel. The retention of nutritive enemata depends largely upon the gentleness with which they are administered. The tube must be withdrawn most carefully.

3. *Therapeutic.*—Many drugs can be given by rectum. The dose is commonly twice that given by mouth. Bromides, chloral, paraldehyde, and other disagreeable sedatives may be given in this manner. Stimulants such as whiskey and coffee, simple saline solution, digitalis and the like may be likewise administered. In giving such enemata the irritating qualities of the substance to be given must be borne in mind,

and dilution with water or normal salt solution practiced accordingly. The total quantity should not, however, exceed six or eight ounces.

COLON IRRIGATION. LAVAGE OF THE COLON.—In dysentery, typhoid fever, colitis of various forms, and also in various types of intoxication, anemia, chronic rheumatism, and the like, the irrigation of the colon with large quantities of warm normal salt solution may be employed. The solution at a temperature of about 110° F. is held in a large douche-bag or other reservoir. A double rectal tube (Kemp's) with one channel for the entrance and another for the outflow of the fluid is commonly employed. With this apparatus from one to two gallons of warm salt solution is allowed to flow slowly in and out of the bowel in the course of from 30 minutes to an hour. The patient lies ordinarily upon the back or the left side. The knee-chest position is recommended if it is desired to carry the fluid as high as possible in the colon, but it is too trying for general use.

Instead of the double tube a single rectal tube with a "T" attachment may be employed, the solution being run in through one arm of the "T" till the bowel is moderately distended and then allowed to escape through the other arm, which is made to empty into a receiving vessel.

ENTEROCLYSIS.—The more slowly the solution flows in and out the more of it will be absorbed. Murphy, of Chicago, has recently suggested the advantage of allowing salt solution to drip into the bowel continuously for hours with the object of its slow but steady absorption. If it is desired to hasten the process the reservoir is suspended just sufficiently above the level of the patient's body to cause the fluid to flow very slowly into the rectum. In this way about a pint of normal salt solution may be given and absorbed in an hour.

HYPODERMOCLYSIS.—Normal salt solution to the amount of 500 c.c. to 1,000 c.c. may be injected into the soft cellular tissues of the skin, and thence be gradually absorbed into the circulation. An ordinary douche-bag armed with an aspirating needle may serve the purpose, but in most hospitals more satisfactory apparatus is in use. The salt solution is sterilized in flasks holding 1,000 c.c. The flask is placed at the head of the bed about 3 feet above the level of the patient's body. From the flask rubber tubing, weighted at the end to keep it in the flask, leads to the clysis needles. Near the end a glass "Y" is inserted to either arm of which one of the needles is attached. The ordinary aspirating needles may be used, but the finer the needle the slower will be the flow of the solution and the less traumatism will be done. In emergencies it may be desirable to introduce the fluid as rapidly as may be. In most cases, however, it is best to make the injection as slowly as possible, an hour or two being occupied in the administration of 1,000 c.c. If fine needles are used, the patients may be quite undisturbed by the procedure, and the injections may be frequently repeated. The usual sites chosen are the pectoral or lateral thoracic regions, but the injec-

tions may be made into any of the loose cellular tissue.   Perfect asepsis must be practiced as to the fluid, apparatus and the hands of the operator.   Care should also be taken to exclude air from the tubing before beginning the injection.   The sites of puncture are to be protected by pads of sterile gauze.

INTRAVENOUS INFUSION OF NORMAL SALT SOLUTION.—The solution may be introduced directly into a vein, usually the median basilic or median cephalic vein.   For this purpose a ligature is bound about the arm to make the veins stand out.   Usually the selected vein is then exposed and two ligatures passed under it close to one another and left untied.   The vein is then nicked between the ligatures on one side and into the opening a special small canula connected by tubing to the flask of salt solution as for hypodermoclysis is introduced. One of the ligatures then secures the canula in the vein, while the other ties off the vein below.   As soon as the canula is safely in the vein the ligature on the arm is cut, and the solution is allowed to flow slowly into the vein. Here again it is desirable to make the injection slowly, that the heart may not be embarrassed by too sudden an increase of its contents.

A much simpler method will ordinarily answer the purpose.   The arm is ligatured as before, and when the veins are well distended a small aspirating needle is thrust obliquely into one of them.   As soon as a free flow of blood from the needle indicates that it is in the vein, the rubber tubing leading from the flask is slipped over the end of it and the fluid is allowed to flow as before.   It is, of course, necessary to start the flow of the solution through the tubing before connecting it to the needle.   After the procedure the seat of puncture should be protected by a pad of sterile gauze.

BLEEDING.—The withdrawal of blood from the arm is usually effected in the ancient manner of constricting the upper arm and then opening the median basilic or median cephalic veins at the elbow.   From 8 to 16 ounces of blood are withdrawn.

Blood may be drawn from one of these veins by an aspirating apparatus.   To prevent the clotting of the blood in the tubes or needle sterile liquid albolene is first drawn through them.   The aspirating needle is plunged obliquely into the distended vein exactly as for an intravenous injection.   Perfect asepsis must be observed in either procedure.

BATHS.—*Cold Sponge.*—Water at ordinary temperature, 65° to 70° F., may be used or the temperature may be raised or lowered as desired, the colder the water, naturally the more effective it will be in reducing temperature.   The patient's loins are covered by pinning a towel around him.   The bed is protected by rubber sheeting.   The patient is then uncovered and sponged from head to feet, front and back.   The sponging should alternate with gentle friction of the skin by the nurse's hand.   For this reason it is well to have two nurses.   Such a bath is usually given for 15 or 20 minutes.

Alcohol Sponge.—Alcohol in varying proportions is added to the

water for this purpose. From 25 to 50 per cent. is commonly used. The more rapid evaporation of the alcohol intensifies the effect of the bath, and its addition is usually grateful to the patient. Whenever such a bath is given care should be taken to wet the head, or a cold wet cloth should be kept upon the forehead.

TUB BATHS.—*Cold.*—The Brandt method is described on page 330.

Hot.—For a hot bath it is best to fill the tub with water at a temperature of 90° to 95° F., and after the patient is immersed further raise the temperature by adding more hot water. In this way the temperature may be raised to 105° to 110° F. The head should be constantly wet with cool water. Such a bath may be continued for 15 or 20 minutes. On leaving it the patient may be swathed in blankets and the perspiration induced by the bath thus protracted for half an hour. When only a sedative effect is sought from such a bath, the temperature should not exceed 100° F.

HOT-AIR BATH.—The bed is protected by rubber sheeting, covered by a blanket on which the patient lies. The body of the patient is covered by bed-cradles supporting several layers of blankets, which are tucked closely about the neck and under the patient's feet. The hot-air apparatus ordinarily consists of a length of small-sized stove-pipe, large enough at one end to hold an alcohol lamp and tapering toward the other end, which is also bent at an angle so as to render it easy to introduce it under the bed-clothing at the foot of the bed. The portion of the pipe likely to be touched by the blankets must be protected by asbestos or wet cloths. When the bed has been arranged to make the air-chamber about the patient's body as tight as possible, the alcohol lamp is lighted and placed in the lower end of the pipe through a trap-door provided for the purpose, and the door closed. The air above the patient should reach a temperature of 150° to 175° F. The head must be kept cool by an ice-cap or cold wet cloth. Unless fluids are restricted, water, hot tea or lemonade may be given during the bath, which should be continued for 20 minutes or more after the desired temperature has been reached. The bath over, the patient should be rubbed dry, rolled in dry blankets, and allowed to lie quiet for an hour, with the ice-cap to his head and a hot-water bottle at the feet.

At the end of this time the body is rubbed with alcohol and the bath is over.

Cabinet-baths.—Portable cabinets arranged to fit closely about the neck of a patient and surround the body as he sits in a chair, are commonly employed. The heat is supplied by an alcohol lamp or gas-burner placed under the chair.

Electric-light cabinets in which the heat is supplied by numbers of incandescent lights are also employed for this purpose. They are safe and very easily managed.

THE HOT PACK.—*Dry.*—The patient is wrapped in several layers of blankets with hot-water bottles at the feet, between the legs and in

each axilla. Instead of the hot-water bags bricks heated in an oven may be used. Care must be taken to wrap bags or bricks in such a way as to prevent burning the patient. The head is cooled as in the hot-air bath and hot drinks may be given. The pack is continued for half an hour, and then terminated like a hot-air bath.

Wet.—For the wet hot pack blankets wrung out of water heated to 150° F. are used. The patient is swathed in these, and hot-water bottles placed as for the dry pack. The head must likewise be kept cool, and the hot drinks may be given. The pack should be continued about one-half an hour, and then terminated like a hot-air bath.

Cold Pack.—With the bed protected, the patient is wrapped in a sheet wet in cold water of any desired temperature, and the sheet is frequently sprinkled with more water during the course of half an hour in which the patient remains in the pack. Throughout the process vigorous rubbing is kept up through the sheet. When the pack is ended the body should be rubbed thoroughly dry.

Local Hot or Cold Applications.—Priessnitz compress. The chest, abdomen or any of the extremities may be treated by compresses wrung out of water either hot or cold, as may be desired. The compresses must be frequently changed to maintain as nearly as possible the temperature desired.

Mustard Paste.—Mustard and flour are mixed usually in the proportion of 1 to 4 or 6 for an adult, and then stirred into a paste with a little cold water. The paste is spread on gauze or cheesecloth of any desired size, and applied to the surface of the body. The other surface should be covered by more gauze or linen. Mixing the white of an egg or a little oil with the paste is said to lessen the risk of blistering. The action of the paste on the skin must be frequently observed, and the application removed when the skin is well reddened. The area treated should then be washed with warm soap and water to remove any of the application still adhering to the skin, and, if very red, anointed with vaseline.

Flaxseed Poultice.—To about a pint of boiling water enough flaxseed meal is slowly stirred in to make a paste just thin enough to drop from a spatula or spoon. Thorough beating of the mixture is said to make it "lighter." The hot meal is then spread on gauze or cheesecloth, the reverse side covered by more gauze and applied to the desired surface of the body. A further covering of oiled silk serves to retain the heat. Such a poultice will keep hot for about an hour, and should then be removed. The reddened area is covered by cotton.

As a matter of convenience the paste known as cataplasma kaolini may be used instead of the flaxseed.

Ice Coil.—The coil consists of small-sized rubber tubing (7 mm. in diameter) fastened in a coil about 25 cm. diameter. One end of the coil is led up to a tub of ice-water several feet above the level of the patient's body, the other end empties in a receiving pail. The water

is started through the coil by suction. The coil is commonly applied to the abdomen, but may be used on the head. When working perfectly it is very effective in reducing the local temperature, but it must be constantly watched to be sure that the flow has not been stopped by some accidental kinking of the tubing.

STANDARD DIETS

The amount of food necessary varies greatly under different conditions of health, and still more markedly in disease. Age and weight, bodily activity and climate have important influence upon it. It undoubtedly varies in different individuals under apparently like conditions. For a man at rest it is calculated that the daily ration should contain approximately 35 calories per kilogram of body weight. For a child one year old the ratio is 90 calories per kilo; at two years, 80; at ten years, 60, and at fourteen years, 52. On this basis it is estimated an adult of about 150 pounds weight (70 kilograms), resting, would require a daily ration representing about 2,500 calories.

The distribution of these values between the several food principles is a subject much studied and discussed. Voit's standard allowed 100 to 120 grams of protein per day. Chittenden finds that healthy men may require only 40 to 60 grams of protein. Other calculations exceed Voit's standard. As an average one may say that the 2,500 calories may be drawn from 100 grams of protein, 50 grams of fat, and 400 grams of carbohydrates.

In febrile conditions, especially in typhoid fever, there is a rapid loss of weight, ascribed to destruction of the protein of the body as a result of toxemia. To meet this loss it is calculated that the typhoid-fever patient requires 40 calories per kilo., or for the patient of 70 kilograms 2,800 to 3,000 calories. Efforts to bring the typhoid dietary up to this standard by feeding cream, lactose and other carbohydrates freely have apparently been advantageous. How generally this example may be followed or with what advantage is not yet known.

In most hospitals we find in use a series of diets which under ordinary conditions are employed in the treatment of the sick. These, for convenience, are designated as fluid diet, soft diet, convalescent diet.

Fluid Diet.—Milk, beef tea, chicken broth, mutton broth, egg albumin; 6 to 8 ounces of one or the other every 2 or 3 hours.

Soft Diet.—Gruels, junket, custard, cereals, eggs (boiled or poached), milk toast, farinaceous puddings, tea, coffee, milk. These feedings may be given every 2 or 3 hours still, or grouped in three meals with lighter feedings between.

Convalescent Diet.—This is regularly arranged in three meals, as follows:

Breakfast.—Tea or coffee with milk or cream and sugar. Bread and butter—white bread or graham bread, or cornbread, or rolls, or

toast. Porridge: hominy, farina or other cereal. Meats: eggs, fish, bacon.

DINNER.—Soup—stock, or chicken, or mutton broth with barley or vegetable. Bread and butter. Meats—beef, roast or broiled. Chicken or fish. Vegetables—potatoes, mashed or baked, rice, macaroni, hominy, peas, beans (string), spinach, etc. Puddings, rice, bread, farina, tapioca, corn starch or custard.

SUPPER.—Tea with cream or milk, and sugar. Bread and milk, milk toast, bread and butter, toast and butter. Cooked fruits, such as apples, stewed or baked, or as apple sauce, stewed prunes or pears.

To the supper any of the articles enumerated under soft diet are added when necessary.

Low Salt or Salt-free Diet.—The quantity of salt in any of these diets may be reduced to a minimum by withholding salt in the preparation of the food and allowing no salt with the meals. In each instance a small quantity of sodium chloride naturally present in the several foods remains. For the majority of cases the salt diet thus modified is employed when such a reduction of the salt intake is desired.

Under most conditions the fluid, soft and convalescent diets are ordered in succession with no thought to the quantity of food taken or the value of the diet as a whole. The acutely sick patient is ordered a fluid diet, and with the return of appetite, which usually accompanies the subsidence of fever or pain, the soft diet is given, and later the convalescent. In cases of protracted illness it may be of great importance to be able to calculate the amount and the value of the food taken and to determine its sufficiency.

Under the supervision of Dr. H. S. Carter the effort has been made to learn the appropriate composition and food value of the food actually received by the patients. For this purpose a sample diet of each class, that is, the equivalent of a diet actually served in the wards in the course of the regular work, was taken, the amounts of the several foods determined, the whole mixed together, dried in bulk, reduced to a powder, and then analyzed for the chief constituents with the following results:

FLUID DIET

Quantities Given

	gms.
Coffee	164
Broth	676
Gruel (Oatmeal)	202
Tea	200
Milk	600
Sugar	67
Lemon juice	35
Egg albumin	92
	2036

This diet is served in quantities of about 6 ounces (180 gms.) every two hours. As the patients are allowed considerable choice the amounts of the several foods will vary in different cases. The sugar and part of the milk are given with the coffee and tea, the lemon juice with the egg albumin.

SOFT DIET
Quantities Given

Breakfast	*Gms.*	*Dinner*	*Gms.*	*Supper*	*Gms.*
Toast...........	12	Broth..............	165	Bread.............	30
Bread............	41	Bread.............	12	Toast.............	30
Sugar............	19			Farina............	91
Oatmeal..........	83			Butter............	8
Milk.............	215	Potato with skin....	95	Apple sauce.........	133
Coffee...........	170	Rice boiled.........	107	Sugar.............	12
Butter...........	6	Corn starch.........	80	Tea..............	180
Egg (no shell)......	45	Ice cream..........	42	Milk.............	210
	591		501		704

CONVALESCENT DIET
Quantities Given

Breakfast	*Gms.*	*Dinner*	*Gms.*	*Supper*	*Gms.*
Lean meat..........	33	Lamb.............	88	Milk.............	170
Corn Bread.........	45	String beans........	37	Tea..............	180
Toast............	20	Boiled rice.........	80	Bread............	29
Sugar............	26	Cup custard........	124	Cup custard (sweetened)	71
Oatmeal (cooked).....	55	Bread............	18	Farina............	85
Butter............	5	Vegetable soup......	200	Cooked prunes (little sweetened).........	98
Milk.............	200	Tapioca pudding (sweetened)............	63	Sugar............	33
Coffee...........	200	Potato with skin (baked) 20 gms.....	163	Butter............	17
	594		773		683

SALT-FREE DIET (SOFT)
Quantities Given

Breakfast	*Gms.*	*Dinner*	*Gms.*	*Supper*	*Gms.*
Bread............	28	Sugar............	11	Egg (1)...........	29
Sugar............	11	Bread............	28	Toast............	14
Farina...........	55	Butter............	19	Bread............	22
Butter...........	26	Rice.............	69	Butter............	15
Egg (1)..........	29	Farina...........	106	Custard (sugar 15)....	102
Coffee...........	150	Tea..............	160	Prunes (sugar 15).....	63
				Tea..............	190
	299		393		435

ANALYSES OF DIETS

	Fluid Diet	Soft Diet	Convalescent Diet	Salt Free Soft Diet
	Gms.	*Gms.*	*Gms.*	*Gms.*
Total weight.................	2045.5	1756.	2054.50	1593.
Moisture....................	1830.	1393.5	1594.50	1317.
Protein (estimated)...........	47.32	49.48	89.35	34.53
Nitrogen (determined)........	7.57	7.91	14.29	5.52
Fat........................	28.01	49.95	70.09	65.16
Carbohydrate................	108.98	291.24	256.02	140.76
Calories....................	898.59	1878.88	2128.42	1257.70
Chlorides...................	2.67	3.36	5.31	.96

Caloric Value of a Diet.—To obtain this accurately we must make such analyses as those above or know the exact amounts of each food consumed and the chemical composition of each. Each gram of fat is equivalent to 9.3 calories, and each gram of protein or carbohydrate to 4.1 calories. In ordinary practice it is difficult to carry out these procedures, but the value of the diet can be obtained with sufficient accuracy by the aid of such a table as the following:

TABLE OF FOOD-VALUES IN UNITS OF 100 CALORIES

Milk, 5 oz.	5.
Cream, 16 per cent. (2 oz.)	1.5
Beef tea, 2½ pints	10.0
Gruels, without milk, 10 oz.	4.0
Buttermilk, one and one-half glasses (9.5 oz.)	8.
Koumis, one glass (7 oz.)	5.
Whey, two glasses (13 oz.)	3.5
Eggs, one and one-half	10.
Egg albumin (whites of 2 eggs to 6 ounces water), 3 glasses	24.
Whites of eggs, 6	24.
Yolks of eggs, 2	4.5
Custards, 4 oz.	7.0
Oatmeal, one and one-half serving (5.5 oz.)	4.25
Boiled rice, ordinary cereal dish (3 oz.)	2.5
Hominy, large serving (4.2 oz.)	2.5
White bread, home made, one thick slice (1.25 oz.)	3.2
One small Vienna roll (1.25 oz.)	3.2
Butter, one pat (1.5 oz.)	0.0
Sugar, three teaspoonfuls, one and one-half lumps (0.8 oz.)	0.0
Oil, one-third ounce	0.0
Codfish, two servings (5 oz.)	23.
Halibut steaks, one serving (2.8 oz.)	15.
Mackerel, Spanish, one serving (2 oz.)	12.2
Shad, one serving (2.1 oz.)	11.2
Salmon, small serving (1.5 oz.)	7.3
Oysters, 12	12.
Roast beef, ordinary serving (1.8 oz.)	10.
Small sirloin steak (1.4 oz.)	7.5
Leg of lamb or mutton, ordinary serving (1.8 oz.)	10.
Lamb chop, one small (1 oz.)	6.
Bacon, small serving, medium fat (0.5 oz.)	1.5
Chicken, broiler, edible portion, large serving (3.2 oz.)	19.
Turkey, large serving (1.2 oz.)	7.
Potato, baked, one, good size (3 oz.)	3.75
Potato, sweet, baked, one-half average potato (1.7 oz.)	1.5
String beans, five servings (16.66 oz.)	3.75
Spinach, two ordinary servings (6.1 oz.)	3.7
Peas, green, one serving (3 oz.)	5.7
American or Swiss cheese, 1.5 cubic inches (0.75 oz.)	6.
One baked apple, 3.3 ounces	0.5

TABLE OF EQUIVALENT WEIGHTS AND MEASURES

Grains to Grams		Grams to Grains		Avoirdupois Ounces to Grams		Grams to Avoirdupois Ounces		U.S. Fluidounces to C.C.		C.C. to U.S. Fluidounces	
Grains	Grams	Grams	Grains	Ounces	Grams	Grams	Ounces	Fluid-ounces	Cubic Centimeters	Cubic Centim.	Fluidounces
1	0.0648	1	15.432	1	28.350	1	0.035274	1	29.6	1	0.0338
2	0.1296	2	30.865	2	56.699	2	0.070548	2	59.2	2	0.0676
3	0.1944	3	46.297	3	85.049	3	0.105822	3	88.8	3	0.1014
4	0.2592	4	61.729	4	113.398	4	0.141096	4	118.3	4	0.1352
5	0.3240	5	77.162	5	141.748	5	0.176370	5	147.9	5	0.1690
6	0.3888	6	92.594	6	170.097	6	0.211644	6	177.5	6	0.2028
7	0.4536	7	108.026	7	198.447	7	0.246918	7	207.1	7	0.2366
8	0.5184	8	123.459	8	226.796	8	0.282192	8	236.7	8	0.2704
9	0.5832	9	138.891	9	255.146	9	0.317465	9	266.3	9	0.3042
10	0.6480	10	154.323	10	283.495	10	0.352739	10	295.8	10	0.3380
11	0.7128	11	169.756	11	311.845	11	0.388013	11	325.4	11	0.3718
12	0.7776	12	185.188	12	340.194	12	0.423287	12	355.0	12	0.4056
13	0.8424	13	200.621	13	368.544	13	0.458561	13	384.6	13	0.4394
14	0.9072	14	216.053	14	396.894	14	0.493835	14	414.2	14	0.4732
15	0.9720	15	231.485	15	425.243	15	0.529109	15	443.8	15	0.5070
16	1.0368	16	246.918	16	453.593	16	0.564383	16	473.3	16	0.5408
17	1.1016	17	262.350	17	481.942	17	0.599657	17	502.9	17	0.5746
18	1.1664	18	277.782	18	510.292	18	0.634931	18	532.5	18	0.6084
19	1.2312	19	293.215	19	538.641	19	0.670205	19	562.1	19	0.6422
20	1.2960	20	308.647	20	566.991	20	0.705479	20	591.7	20	0.6760
30	1.944	30	462.970	30	850.486	30	1.05820	30	887.5	30	1.014
40	2.592	40	617.294	40	1133.982	40	1.411	40	1183.4	40	1.352
50	3.2400	50	771.617	50	1417.477	50	1.764	50	1479.2	50	1.690

One pound = 0.453 Kilos.   1 Kilo. = 2.205 pounds   One pint = 473.3 C. C.   One litre = 1.056 quarts or 2.113 pints

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